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1. Goldfarb, A. F., and Napp, E. E.: Use of Methallenestril (Vallestril) in Control of Menopausal Symptoms, J.A.M.A. *161*:616 (June 16) 1956.
2. Schneeberg, N. G.; Perczek, L.; Nodine, J. H., and Perloff, W. H.: Methallenestril, a New Synthetic Estrogen, J.A.M.A. *161*:1062 (July 14) 1956.

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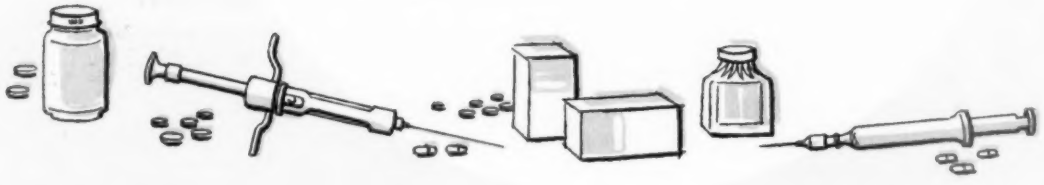

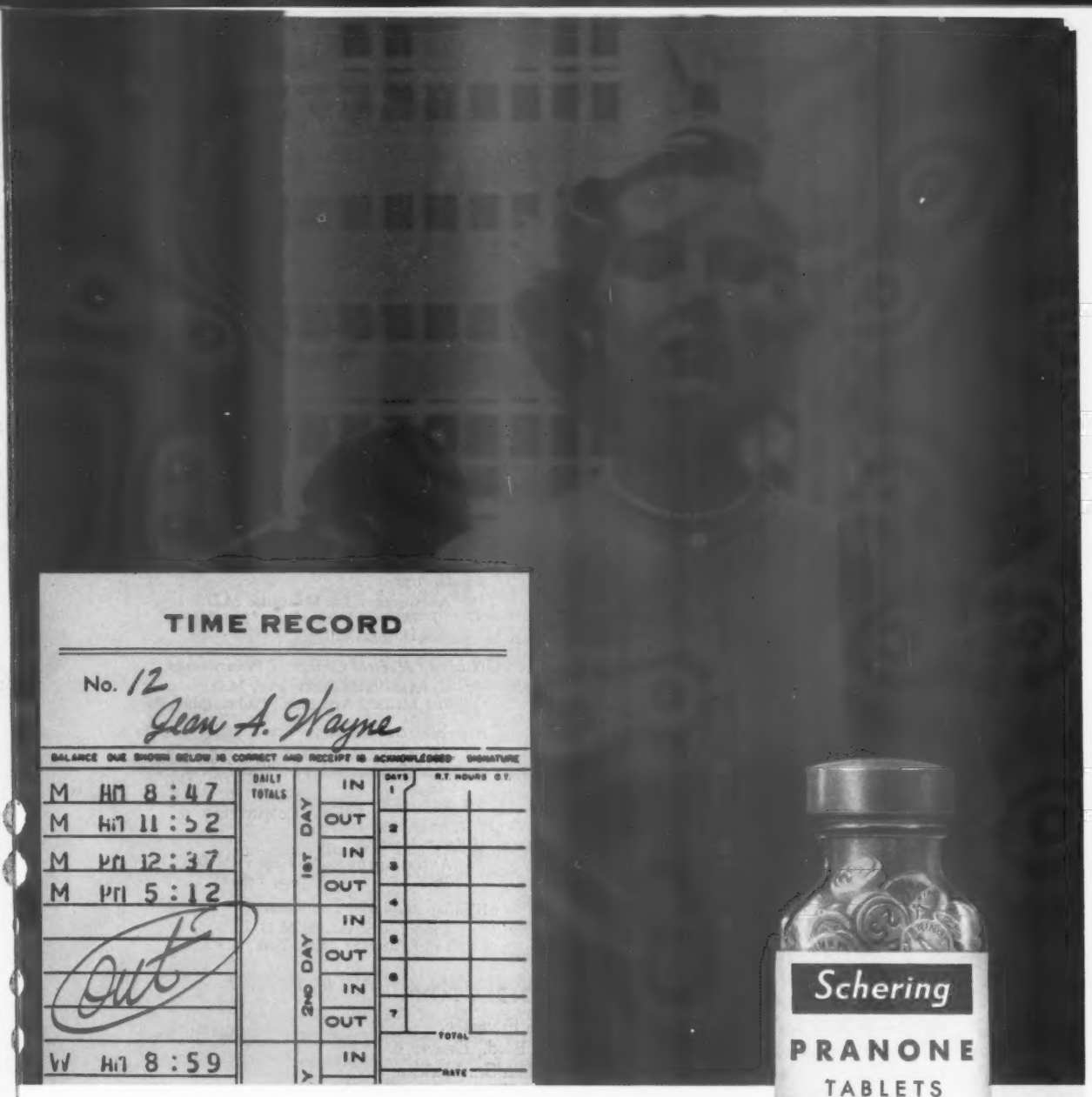

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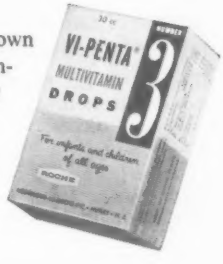

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(Continued on page 17)

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(Continued on page 18)

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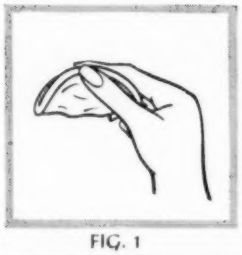


FIG. 1

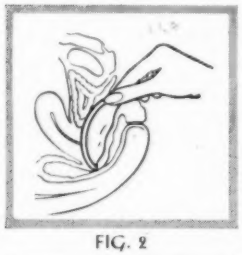
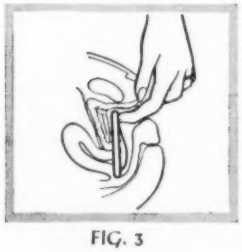


FIG. 2



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References:

1. McLaren, H. C.: The present status of hormone therapy at the menopause. Practitioner *171*:500, Nov. 1953. **2.** Sandes, S. G.: The management of the menopause. M. J. Australia *1*:274, Feb. 20, 1954. **3.** Peterson, P.: Management of the climacteric. Mil. Med. *116*:348, May 1955. **4.** Gold, H.: Management of the menopause, in Cornell Conferences on Therapy, vol. 4, edited by H. Gold, Macmillan, New York, 1951, pp. 301-321. **5.** Adams, R. D.: Disorders of nervous function, in Principles of Internal Medicine, edited by T. R. Harrison, McGraw-Hill, New York, 1954, pp. 227-233. **6.** Donovan, J. C.: Psychologic aspects of the menopause. Obst. & Gynec. *6*:379, Oct. 1955. **7.** Selling, L. S.: A clinical study of Miltown, a new tranquilizing agent. J. Clin. & Exper. Psychopath. *17*:7, March 1956. **8.** Borrus, J. C.: Study of effect of Miltown (2-methyl-2-n-propyl-1,3-propanediol dicarbamate) on psychiatric states. J.A.M.A. *157*:1596, April 30, 1955. **9.** Goodall, J. R.: Premarin in some post-menopausal complications. J. Obst. & Gynaec. Brit. Emp. *49*:660, Dec. 1942. **10.** Hamblen, E. C.: Irregularities of uterine bleeding and their treatment, in M. Clin. North America, edited by W. O. Thompson, Saunders, Philadelphia, Jan. 1948, pp. 57-72. **11.** Shorr, E.: Management of the menopause. Am. J. Med. *10*:91, Jan. 1951.

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It is both effervescent and antacid.

"The emptying time of the stomach is actually shortened by reducing the gastric acidity."[1]

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1. The Physiological Basis of Medical Practice. 1945, p. 486.
2. New England J. Med. 235:80 (July 18) 1946.

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Reversed Passive Rh Sensitization in Guinea Pigs

Ruth Renter Darrow, M.D., and Irene Shmigelsky, M.D.

In our clinical studies of so-called hemolytic disease of the newborn infant we observed certain facts that could not be explained solely on the basis of hemolysis. Among these observations were the following: 1. A positive direct van den Bergh reaction of the umbilical cord blood indicated fetal liver injury in several cases in which there was an absence of jaundice at birth and an icterus index too low to be consistent with intrahepatic obstruction by excessive bilirubin. 2. Correlation between brain injury and significant anemia was frequently lacking. 3. Early transfusions of Rh-positive or Rh subtype-specific blood produced in many cases prompt amelioration of the jaundice, edema, enlargement of liver and spleen, and the erythroblastemia, even though anemia recurred. Furthermore, the fulminating intoxication often seen in icterus gravis neonatorum may be largely absent in congenital anemia, a condition in which the immune reaction produces destruction of red blood cells often more severe and usually no less rapid. Organic involvement resulting from sensitization of tissue in liver and lungs, on the other hand, affords not only an adequate explanation for these findings but also an inescapable consideration. Levine,[1] in discussing the role of isoimmunization by the A and B factors in the production of erythroblastosis fetalis, stated that "one may speculate that fatal effects may result when the reaction takes place in, or upon, vital organs or tissues." Our observations suggest that serious effects likewise occur in Rh sensitization and that they may, at least partially, be prevented by adsorption of antibody globulin on fresh (donor's) Rh-positive erythrocytes.[2]

While at least three differently behaving anti-Rh globulins can be demonstrated by reactions between Rh-positive red blood cells and specific antiserum, [3-11] including that blocking all specific agglutination,[12] tissue-sensitizing antibodies capable of causing an anaphylactic type of reaction have not been demonstrated. The experiments here presented were devised to determine whether such sensitizing antibody could be shown to be present in the serum of Rh-negative mothers who had borne infants affected with erythroblastosis fetalis.

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Experiments

Method. Two groups of guinea pigs were injected intraperitoneally, one with Rh-positive red blood cells and the other with Rh-negative erythrocytes. These two groups in turn were divided into two subgroups, one of which received anti-Rh serum and the other normal serum. Two mothers, each of whom had had two affected children, and whose serum contained blocking antibodies, were the donors of the anti-Rh serum and of the Rh-negative erythrocytes. Normal serum was obtained from Rh-positive individuals who had no history of receiving transfusions. Since the husbands of the

TABLE I
Injection and Reaction in Experimental Group: Guinea Pig Number 12

Rh-Positive Erythrocytes	Normal Serum	Anti-Rh Serum	Reaction*
—	—	4.0 cc.I.P.	—
—	—	4.0 cc.I.P.	—
2 cc.(50%)	—	—	0
—	—	4.0 cc.I.P.	0†
2 cc.	—	—	0
—	—	4.0 cc.I.P.	+++
2 cc.	—	—	0
—	4.0 cc.I.P.	—	0‡
—	—	4.0 cc.I.P.	+++
2 cc.	—	—	0
—	—	4.0 cc.I.P.	+++

I.P.=intraperitoneal injection. Injections of either cells or serum were made daily.

*There is no reaction upon injection of Rh-positive erythrocytes.

†There is no reaction upon injection of specific antiserum following only one injection of erythrocytes, but there is a severe reaction with each injection of antiserum subsequently.

‡There is no reaction upon injection of normal serum after adequate injections of erythrocytes.

Rh-negative mothers were each Rh_1 (CDe), this subtype of Rh-positive cells was used exclusively. Only serum and cells from group O individuals were employed. All cells were washed five times and given intraperitoneally in 2 cc. amounts of 10 to 50 per cent suspensions. Serum was given in 4 cc. amounts when given intraperitoneally and in amounts of approximately 2 cc. when given by vein. Cell suspensions and serums were cultured before using to ensure sterility.

Results. Our early attempts to demonstrate passive anaphylaxis in the usual manner, sensitizing first with the antiserum and then injecting the antigenic erythrocytes, were entirely without results. When, however, these animals were given subsequent injections of antiserum following further administration of Rh-positive erythrocytes, definite reactions took place (table I). Reaction occurred in each instance only when injection of antiserum was made; no significant effects were observed with injection of Rh-positive red blood cels. There was no reaction following antiserum injection when Rh-negative erythrocytes were employed (table II).

When several injections of Rh-positive erythrocytes preceded the first injection of specific antiserum, there was a definite positive reaction: After immediate urination and defecation, the animal rubbed its nose vigorously; then, after a brief period of increased activity, the hind legs seemed to become suddenly weak; retching sometimes occurred; the respiration became first rapid, then slow and labored; and the animal began to "list" to one side, and finally could no longer force himself to return to the upright position and sprawled out with his hind legs behind him or lay helpless on his side. The eyes were dull; muscular twitches progressed to convulsive movements; and defecation became soft and even blood tinged. Death rarely occurred before 30 minutes, and in some cases the animal remained in a state of extreme lethargy with labored breathing until death one to three hours later. In case of death we scored the reaction +4; if the animal ultimately recovered from an extreme reaction, we recorded the result as +3; where the same symptoms were of shorter duration but still presented severe dyspnea, muscular in-co-ordination and weakness, frequent defecation, retching, sneezing, and nasal irritation, we called the reaction +2. Milder symptoms with a tendency in the animal toward leaning or falling to one side, apathy, ruffling, and definite dyspnea, but without labored breathing, were noted as +1 or doubtful (+ —).

EXPERIMENTAL GROUP: The reactions in the experimental group varied from 0 to +4 (table III). Seven reactions out of 43 were negative (16.3 per cent); of these, 4 were in animals that had received only one injection of erythrocytes preceding the injection of antiserum. Two of the negative reactions occurred consecutively in an animal that had reacted positively six times previously (animal 14), a fact that suggests the development in this animal of an active immunity against the antiserum. Thirty of the 43 reactions were +2 or more (69.7 per cent, while 7 of the 16 experimental animals died, a mortality of 43.8 per cent.

CONTROL GROUP: In the control series 58 of 66 reactions (87.9 per cent) were negative. Only the 2 fatal reactions (3.0 per cent) were definitely positive. With two deaths out of 36 animals the mortality in the control series was 5.9 per cent.

COMMENT: Human serum given intravenously is

TABLE II
Injection and Reaction in Control Group: Guinea Pig Number 32

Rh-Negative Erythrocytes	Anti-Rh Serum	Reaction*
—	1.0 cc. I.V.	—
—	2.0 cc. I.P.	—
2 cc. (50%)	—	—
—	4.0 cc. I.P.	—
—	4.0 cc. I.P.	0
—	4.0 cc. I.P.	0
2.2 cc.	—	0
—	4.0 cc. I.P.	0
2 cc.	—	0
—	4.0 cc. I.P.	0
2 cc.	—	0
—	4.0 cc. I.P.	0

I.V.=intravenously; I.P.=intraperitoneally.

*There is a complete absence of reaction.

not always tolerated by guinea pigs. This is indicated in our first trials by the sudden death of 4 animals which occurred with the first injection of serum (not heat treated). Serum from blood drawn less than 48 hours before administration and not heat treated at 56 C., for example, may produce toxic results.[13,14] While most of the serum given to our animals was sufficiently aged or heat treated, we do not know whether the factor of toxicity explains any of the deaths in our series. It may be seen, however, that the percentage of negative reactions in the experimental group, as compared with the control series, was roughly 1:5, whereas the ratio of the percentages of the fatal reactions was approximately 5:1.

Responses indicating more precisely the specificity of the reaction in our guinea pigs were seen in those animals that were treated with several injections of Rh-positive erythrocytes and that failed to react to normal serum but that reacted strongly to the following injection of antiserum (table I). In like manner, animals given injections of Rh-negative red blood cells failed to react to the antiserum but reacted positively to antiserum after subsequent injections of adequate amounts of Rh-positive cells.

The consistent absence of reaction with injection of Rh-positive erythrocytes, especially after several injections of specific antiserum indicates that the sensitization reaction does not involve the intact Rh-positive red blood cells. When, however, the Rh-positive blood cells were given first in three daily injections of the 50 per cent suspension, a definite, frequently fatal, reaction occurred in every animal so treated on the first administration of the antiserum (table IV-A). As in the Rh-positive baby, it may be assumed that the Rh-positive erythrocytes are to a great extent disintegrated within the phagocytic cells. Since in our animals the erythrocytes were injected into the peritoneal cavity, the Rh lipoprotein released by the phagocytic action of the Kupffer cells and by the erythrophagocytic cells of the spleen would find its way in large part into the liver cells along with the iron and pigment of the destroyed erythrocytes. The liver would thus be a "shock organ" producing an anaphylactic response of the "dog type" upon injection of the antiserum, as well as the typical

TABLE III
Summary of Results

Reaction	0	±	+	++	+++	++++	Deaths	
							No.	%
Rh-positive blood cells Anti-Rh serum (16 animals)	P_1 P_1 P_1 P_1 P– P# P–	P P V	P P V	PP PP V PP V PP V PP	PP PP V PP V P V	P VV VV VV	—	—
% reactions for experimental group (43)	16.3	7	7	30.2	23.2	16.3	7	43.8
Rh-positive blood cells Normal serum (10 animals)	PP V PP V P	V V	– – –	— — —	— — —	— — —	0	0
Rh-negative blood cells Anti-Rh serum (15 animals)	PPP VVV PPP VVV PPP VVV PPP V PPP	V V	– – – – –	— — — — —	— — — — —	— — V — —	1	6.7
Rh-negative blood cells Normal serum (11 animals)	PPP VVV PPP VVV PPP VVV PPP PP	V	– – – – –	— — — — —	— — — — —	— V — — —	1	9.1
% reactions for control group (66)	87.9	9.1				3.0	2	5.9

V=intravenous injection of serum. P=intraperitoneal injection of serum. P_1=preceded by only one injection of erythrocytes. P'=had reacted previously six times. Apparently immune (14%). P#=reacted subsequently to injections of serum after injections of erythrocytes (26%).

TABLE IV

Specificity of the Reaction

Group (No. of Animals)	Rh-Positive Blood Cells	Rh-Negative Blood Cells	Normal Serum	Antiserum	Reaction
A (19)	2 cc. (50%)	—	—	—	—
	2 cc.	—	—	—	—
	2 cc.	—	—	—	—
	—	—	—	1.7 cc I.V. 2.3 cc. I.P.	++++
B (22)	2 cc. (50%)	—	—	—	—
	2 cc.	—	—	—	—
	2 cc.	—	—	—	—
	—	—	1.7 cc. I.V. 2.5 cc. I.P.	—	0
C (28)	—	2 cc. (50%)	—	—	—
	—	2 cc.	—	—	—
	—	2 cc.	—	—	—
	—	—	—	2.0 cc. I.V.	0

I.V.=intravenously; I.P.=intraperitoneally.

A=experimental group, showing a strong specific reaction with injection of the antiserum after 3 daily injections of Rh-positive erythrocytes. B and C=control groups with no reaction, due to absence of specificity.

respiratory effects usually seen in the guinea pig with anaphylaxis. Normal serum, it may be noted, caused no reaction (table IV-B).

While three injections of Rh-positive erythrocytes apparently provided adequate antigen, one injection seemed not to produce sufficient antigen for the tissues in most instances to allow a reaction with the antiserum injected the following day (table I). When, however, several intravenous injections of antiserum were given first, a faster release of antigen from the subsequently injected dose of Rh-positive blood cells seemed to result, so that a marked reaction occurred in some instances with the subsequently injected antiserum (table V). Such reactions did not occur when Rh-negative erythrocytes were employed in the same manner (tables IV-C and VI). Our data, therefore, suggest that (1) the passive anaphylactic reaction required in the animals' tissues adequate Rh antigen previously released from the red blood cells and that (2) the anaphylactic response occurred as the result of reaction between this Rh antigen and the antiserum, and not between the Rh-positive erythrocytes and antiserum (see Fig. 1).

TABLE V

Reversed Passive Rh Anaphylaxis

No. of Animals	Rh-Positive Blood Cells	Anti-Rh Serum	Reaction*
35	—	2.0 cc. I.V.	—
	—	2.0 cc. I.V.	—
	—	2.0 cc. I.V.	—
	2 cc. (50%)	—	0
	—	2.0 cc. I.V.	++++
64	2.cc. (50%)	—	—
	2 cc.	—	—
	2 cc.	—	—
	—	2.0 cc. I.V.	++++

I.V.=intravenously.

*1. Reaction occurs after one injection of erythrocytes in a sufficient amount to effect rapid red blood cell disintegration, if preceded by antiserum given intravenously. 2. Reaction occurs with the first injection of anti-serum, if preceded by sufficient amounts of erythrocytes, the disintegration of which provides adequate antigen for reaction. 3. Reaction occurs only with the injection of antiserum. Erythrocyte injection causes no reaction.

TABLE VI

Mixed Reactions: Guinea Pig Number 34

Rh-Positive Blood Cells	Rh-Negative Blood Cells	Anti-Rh Serum	Reaction*
—	—	2.0 cc. I.V.	—
—	—	2.0 cc. I.V.	—
—	—	4.0 cc. I.P.	—
—	2 cc. (50%)	—	—
—	—	4.0 cc. I.P.	
2.2 cc. (50%)	—	—	—
—	—	2.0 cc. I.V.	++
2 cc.	—	—	—
—	—	4.0 cc. I.P.	+++
2 cc.	—	—	—
—	—	4.0 cc. I.P.	+++
—	—	4.0 cc. I.P.	0†
—	2 cc.	—	—
—	—	4.0 cc. I.P.	0

*No reaction with antiserum following injection of Rh-negative blood cells.

†The reaction with antiserum following injection of Rh-positive cells is not due to the development of sensitization to serum, since after the absorption of antigen the reaction to serum is negative.

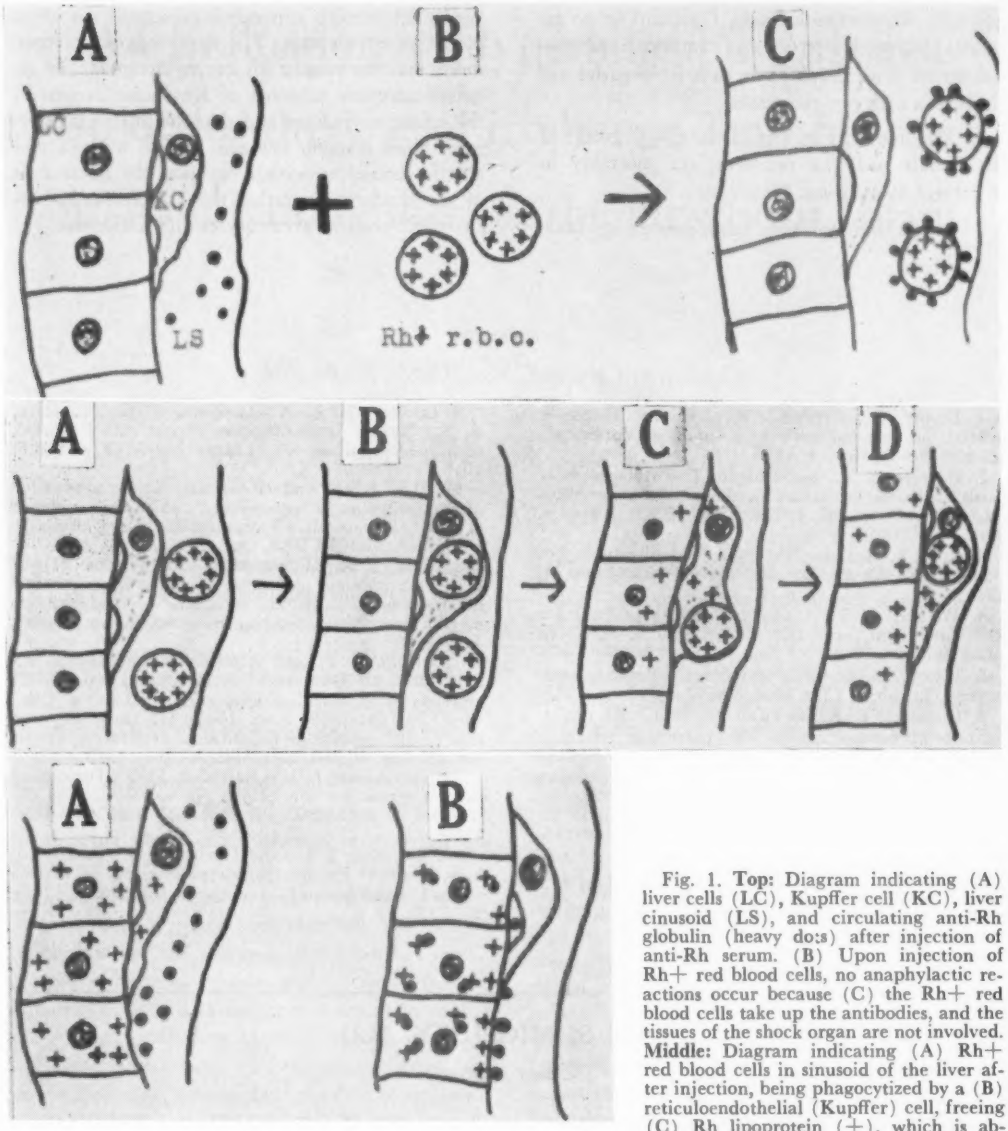

Fig. 1. **Top:** Diagram indicating (A) liver cells (LC), Kupffer cell (KC), liver cinusoid (LS), and circulating anti-Rh globulin (heavy dots) after injection of anti-Rh serum. (B) Upon injection of Rh+ red blood cells, no anaphylactic reactions occur because (C) the Rh+ red blood cells take up the antibodies, and the tissues of the shock organ are not involved. **Middle:** Diagram indicating (A) Rh+ red blood cells in sinusoid of the liver after injection, being phagocytized by a (B) reticuloendothelial (Kupffer) cell, freeing (C) Rh lipoprotein (+), which is absorbed by the (D) hepatic cells along with the pigment and iron of the red blood cells. **Bottom:** Diagram indicating result of injection of anti-Rh serum after preparation with (A) Rh+ red blood cells: Specific anaphylactic reaction (B) occurs between Rh lipoprotein (+) and antibody (heavy black dots) upon or within the tissues of the shock organ, resulting in tissue injury.

Summary and Conclusions

Experiments to demonstrate whether passive anaphylaxis could be produced in guinea pigs with Rh-positive (CDe) human erythrocytes and antiserum from sensitized Rh-negative mothers yielded the following results:

1. No significant reaction occurred with the intraperitoneal injection of erythrocytes at any time before or after injections of antiserum.

2. Reversed passive anaphylaxis was demonstrated by the following reactions: A. Two to 4+ reactions followed the injection of antiserum after injections of Rh-positive erythrocytes in 69.7 per cent of our animals. B. Doubtful or no reactions followed injections of normal serum after prepara-

tion with Rh-positive cells. C. Doubtful or no reactions followed injections of antiserum or normal serum after preparation with Rh-negative red blood cells with two exceptions.

3. The mortality in the experimental group of 16 animals was 43.8 per cent; the mortality in 36 control animals was 5.9 per cent.

The data indicate that tissue-sensitizing (anaphylactic) anti-Rh antibody is present in the serum of sensitized mothers. The data suggest, furthermore, that the passive Rh anaphylactic reaction requires adequate amounts of Rh factor present in the tissues as antigen and that the anaphylaxis results from reaction between the Rh antigen and specific antiserum within or upon the tissue cells of the shock organ rather than from reaction between Rh-positive erythrocytes and antiserum.

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IRENE SHMIGELSKY, M.D.

Guest Editor

Dr. Irene Shmigelsky, Guest Editor of the Branch Two issue of the JOURNAL, is a graduate of the University of Illinois Medical School. She interned at Women and Children's Hospital of Chicago, and had pediatrics training at Children's Memorial and Municipal Contagious Disease hospitals, also of Chicago.

Dr. Shmigelsky first became interested in erythroblastosis fetalis during her internship, when she and a fellow intern, Dr. Josephine Chapin, successfully treated the second of Dr. Ruth Renter Darrow's children to be afflicted with the disease. Continuing interest was fostered later during internship by the teachings of Dr. Darrow herself, who was then "on service" at Women and Children's Hospital. Dr. Shmigelsky subsequently assisted Dr. Darrow in research in the field of Rh at Presbyterian Hospital, on a grant provided through Dr. H. N. Sanford, then Head of the Pediatrics Department at that hospital.

Dr. Shmigelsky is now practicing pediatrics in Park Ridge, Ill., and is a member of the pediatric staff at Presbyterian-St. Luke's Hospital in Chicago.

An Evaluation of Hyperbilirubinemia as a Guide for the Use of Exchange Transfusions in Hemolytic Disease in the Newborn Infant

Mila Pierce, M.D., and Eustacia Rigor, M.D.

THE CURRENT INTEREST in the pathological effects of hyperbilirubinemia in newborn infants was stimulated by Diamond's[1] observations that exchange transfusions reduced the incidence of kernicterus in hemolytic disease of the newborn infant. The superiority of the exchange type of transfusion over simple transfusion in the treatment of hemolytic disease due to maternal isoimmunization has been proved by numerous surveys[2,3] in the past few years. However, the optimum results of this therapy in terms of reduction of the mortality rate in the newborn period, and also in terms of prevention of late neurological sequelae, have been obtained only when the objective of the therapeutic regimen has been to remove the products of hemolysis from the infant's circulation. The exchange transfusion must be completed according to the indication of (1) progressive anemia and (2) the accumulation of the products of hemoglobin metabolism in the plasma.

The case material in the series was drawn from consecutive cases of hemolytic disease of newborn infants, born during 1955 and 1956 at the Chicago Lying-in Hospital of the University of Chicago.

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The necessity of precise criteria for the indication of exchange transfusion has stimulated a vast amount of investigation, the results of which contribute greatly to the practical problems of management of the infant with erythroblastosis. At the same time, new questions have been raised regarding the general problem of jaundice in the newborn infant. A brief discussion of the recent investigations in this field will be attempted in the following discussion.

The level of serum bilirubin in the newborn infant is hyperbilirubinemic by adult standards. The more recent figures collected from data on full-term and premature infants confirm the findings of early investigators. Hsia[4] reported bilirubin levels at birth and throughout the newborn period in four groups of infants: (1) in full-term infants with blood groups compatible with that of the mother; (2) in full-term infants with blood groups incompatible with the mother's; (3) in a group of premature infants; and (4) in a group of infants with erythroblastosis fetalis. In the 12 normal infants whose blood groups were compatible with the mother's, the cord blood levels ranged from 1.4 to 3.2 mg. per 100 cc.; the maximum elevation during the newborn period occurred on the second day, varying considerably in different infants from 3.1 to 12.4 mg. per 100 cc. In 12 normal infants whose blood groups were incompatible with the mother's, the bilirubin levels throughout the neonatal period were similar to those obtained in the "compatible" group of infants. In the premature infants, the levels at birth were similar to those obtained in the cord blood of the full-term infants; on the second day of life the serum level

was equal to that of the full-term infants, but on the fourth day the levels were higher, with a maximum average of 11.2 mg. and a range of 0.3 to 27 mg. per 100 cc. In the premature infant, the hyperbilirubinemia also persists longer than in the full-term infant.

Clinicians have long been aware that a critical level of serum bilirubin beyond which clinical icterus develops in the newborn infant does not exist. Findlay[5] reaffirmed the lack of a critical level in his study of 101 infants; 65 per cent of the infants with serum bilirubin levels in cord blood greater than 1.5 mg. per 100 cc., 45 per cent of the infants with level of 1.0 to 1.5 mg. per 100 cc., and less than 18 per cent of the infants with bilirubin levels less than 1 mg. per 100 cc. developed icterus neonatorum. The icteric color of the skin is an unreliable criterion for measuring the degree or duration of hyperbilirubinemia.

The weight of evidence from numerous studies of icterus neonatorum indicates that ability of the liver to excrete bilirubin is less efficient at birth than it is in the late neonatal period. In his classical study of icterus neonatorum Yllpö[6] found that the amount of bilirubin excreted in the first meconium stool was less in babies who developed icterus than in the nonjaundiced infant. Fashena[7] confirmed these observations and demonstrated a correlation between the maximum serum bilirubin levels in the newborn period and the excretion of bilirubin in the meconium. This finding, which has been well substantiated by other investigators, provides the strongest argument in favor of the inefficiency of the excretory function of the liver of the newborn infant for bilirubin as the dominant cause of icterus neonatorum. Tests for liver function employing injections of bilirubin[8,9] have yielded results that support the theory of hepatic immaturity, although these studies are open to differences in interpretation. The rapid fall in serum bilirubin levels after the third day of life is explained by the improvement in hepatic function that occurs after the circulatory changes coincident with birth. The predisposition of the premature infant to neonatal jaundice is best explained by the theory of hepatic immaturity.

The sole origin of bilirubin is hemoglobin, and one of the most elusive problems in the newborn physiology is the role that hemoglobin metabolism plays in the development of icterus neonatorum. Normally, hemoglobin metabolism begins only after fragmentation of the erythrocytes has reduced the cell to dustlike particles of red blood cell stroma. The hemoglobin is then released and split into the iron containing heme and globulin fractions. The heme breaks down into hemochromogens and bilirubin; the bilirubin passes to the liver and out through the bile into the intestinal tract where it is further metabolized to urobilinogen.

The theory of acceleration of erythrocytic destruction, occurring incident to birth and as a result of readjustment to extra uterine oxygenation, has received little support by modern studies. In studies utilizing transfusions of isotopically tagged erythrocytes obtained from cord blood, Mollison[10] was not able to demonstrate a difference in the rate of destruction of cells in the fetus as compared with the rate of destruction in the adult's cells. In the normal newborn infant the rate of fall in hemoglobin and erythrocytes is greater in the first 2 days of life than in the following 5 to 7 days but not as great as during the second week of life. The amount of hemoglobin released does not permit a direct correlation with the degree of hyperbilirubinemia in the first week of life.

In hemolytic disease of the newborn infant the hemolytic process so far exceeds the limits of the excretory function of the liver that the correlation is apparent. The relationship between the degree of blood destruction as measured by hemoglobin values and the serum bilirubin levels is not a direct one in individual infants. In newborn infants with hemolytic disease, from different centers in England, Mollison and Cutbush[3] found that hemoglobin levels in cord blood were a rough indicator of the severity of the disease. The mortality rate in the newborn period correlated with low hemoglobin levels in the cord blood. He suggested that, since the majority of these deaths were due to kernicterus, a rise in the serum bilirubin levels above 18 mg. per 100 cc. in the newborn period might be a critical level beyond which encephalopathy would occur. With the large amount of clinical experience accumulated since this prophetic suggestion, the critical level of about 20 mg. per 100 cc. of serum bilirubin has retained its ominous significance.

Hsia and his associates[11] found that a single exchange transfusion did not ensure against the development of kernicterus; they showed that kernicterus did not occur when the serum bilirubin levels remained under 20 mg. per 100 cc. but that its incidence increased in infants showing levels approaching 30 mg. per 100 cc. These findings have provided the standards that are in common use when serum bilirubin levels are used as a guide for initial exchange transfusions and for repeated transfusions in the management of the affected infant.

An active argument exists regarding the essen-

tial role of bilirubin in the pathogenesis of kernicterus. It is known that in a certain acute hemolytic anemia in the adult, heme compounds are detectable which are not present in normal serum. The search for these abnormally appearing products of heme metabolism has been rewarding. Tuttle [12] demonstrated excessive amounts of hematin in jaundiced infants with hemolytic disease. Zetterström and co-workers [13] found that excessive amounts of methemalbumin were detectable. Abelson and Boggs [14,15] have employed the measurements of the plasma pigments rather than the bilirubin levels as a guide for the use of exchange transfusions.

Direct evidence of the toxicity of bilirubin was presented by Day,[16] who demonstrated that crystalline bilirubin reduces the oxygen uptake of homogenates of rat brains in vitro. This inhibition was reversed by cytochrome C. The toxic effect of bilirubin for adult rat brain is reversible, but it appears to be lethal for the brain of the newborn rat. Further, Day found that hematin was toxic for brain and diaphragm tissue homogenates.

These recent investigations indicate that there is reason to be guarded in concluding that bilirubin or any other one product of erythrocyte metabolism is the essential cause of encephalopathy in hemolytic disease. The presence of these substances in excessive amounts may have an additive noxious effect. At the present time, other factors in the newborn infant, such as an immaturity of the enzyme system of the brain or even of liver, or variations in the permeability of the blood-brain barrier, or anoxia secondary to pulmonary complications common in the severe form of the disease, are unknown facets of this problem.

In our program for the management of hemolytic disease of newborn infants at the Chicago Lying-in Hospital, the serum bilirubin levels have been employed as an adjunct to the clinical decision for exchange transfusion for the past several years. This test has proved most useful in timing the need for the first exchange transfusion in infants who appear mildly ill at birth, but who exhibit no signs of a rapidly progressive hemolytic process, and also as an indication for repeating the exchange transfusion in the more severely affected infants. An exchange transfusion has been performed at birth or shortly thereafter in all infants classified as moderately or severely ill at birth, according to the criteria given in the tabulation that follows:

In the infants classified as mildly ill at birth, the need for the transfusion was determined by clinical and chemical assessment of the progress of the disease.

Criteria for Classification of Severity of Disease

No disease

1. A positive Coombs' test in the infant and/or a rising titer of maternal antibodies during pregnancy.

Mildly affected

At birth

1. The above serologic signs of isosensitization.
2. Hemoglobin in cord blood above 13 Gm. or in heel blood of 14 Gm. or more.
3. Pallor, splenomegaly, and normoblastosis.

Later

4. Jaundice appearing within the first 24 hours of life, which deepens.
5. The progressive anemia or icterus is mild but behavior of infant remains good.

Moderately severe disease

At birth

1. Hemoglobin less than 13.5 Gm. but more than 11 Gm.
2. Splenomegaly and hepatomegaly.
3. Excessive normoblastosis.

Later

4. Early signs of deepening jaundice, anemia, purpura, or edema.

Severely affected

At birth

1. Hemoglobin less than 11 Gm. plus the aforementioned signs.
2. Early signs of circulatory or respiratory embarrassment or listleness.

Hydrops fetalis

At birth

1. Marked edema at birth, in addition to the signs indicative of a severely affected infant.

Stillborn

TABLE I

Hemoglobin Levels at Birth and Serial Bilirubin Determinations in 31 Infants with Hemolytic Disease of the Newborn Treated with Exchange Transfusions During 1955 and 1956

Case No.	Incompatibility	Hg. at Birth, Gm.	Cord Blood, Mg./100 Cc.	Serum Bilirubin Levels (Mg./100 Cc.), Age in Hours								Exchange Transfusions, Age in Hours				
				6-12	18	24	36	48	60	72	84	1st	2nd	3rd	4th	5th
23	Rh	12.3	12.0	—	22.3	14.0	21.5	—	28.6	38.4	45.0	4½	18	36	61	87
1	Rh	11.5	8.5	—	—	14.9	13.3	21.5	—	—	—	2	22	48	—	—
2	Rh	9.6	7.3	—	—	12.0	9.1	—	—	—	—	1	21	—	—	—
3	Rh	12.8	6.6	4.5	14.4	13.2	—	8.4	—	—	—	3	29	—	—	—
4	Rh	16	6.4	16.3	7.9	—	—	22.0	—	—	—	10½	45	—	—	—
5	Rh	13.7	6.0	—	3.3	—	—	12.8	19.8	11.2	12.8	1½	56	—	—	—
6	Rh	19	3.8	12.0	7.8	—	26.0	13.4	—	—	—	19	39	—	—	—
31	Rh	17.5	3.7	—	14.3	15.3	—	24.6	26.0	17.0	15.0	19	40½	—	—	—
7	Rh	11.6	3.5	—	8.2	—	11.9	13.5	—	6.3	—	5	46½	—	—	—
34	Rh	17.9	2.8	7.9	—	12.5	18.0	9.6	18.5	18.7	10.6	32½	80	—	—	—
37	Rh	16.0	2.6	7.0	9.8	5.4	19.0	22.0	15.7	20.5	12.1	16	37	69	—	—
15	Rh	—	—	—	23.0	—	16.9	13.6	19.4	7.6	1.9	19	53	—	—	—
16	Rh	16.5	—	5.0	—	10.1	—	19.0	14.9	9.5	—	21	42	—	—	—
43	Rh	11.4	6.9	—	—	—	—	10.8	20.0	19.6	13.7	73	—	—	—	—
25	Rh	14.5	6.1	12.8	—	17.0	20.0	9.0	14.0	12.1	9.2	28	—	—	—	—
26	Rh	11.0	5.7	5.1	—	—	—	2.5	—	—	—	7½	—	—	—	—
27	Rh	16.5	4.5	—	15.0	15.4	13.7	13.2	—	—	—	20	—	—	—	—
28	Rh	15.5	4.3	7.5	9.6	17.0	9.0	13.5	17.8	13.0	—	25	—	—	—	—
8	Rh	11.0	3.5	—	—	—	—	—	—	—	—	3	—	—	—	—
9	Rh	15	3.0	—	—	1.5	—	—	—	—	—	12	—	—	—	—
11	Rh	16.2	2.7	2.4	—	—	10.6	16.3	—	—	—	45	—	—	—	—
12	Rh	20	2.6	—	—	4.4	12.8	20.1	—	—	—	51	—	—	—	—
38	Rh	19.7	2.6	6.7	—	—	14.5	17.0	22.0	10.5	—	51	—	—	—	—
40	Rh	11.0	2.1	7.2	—	—	3.9	3.3	—	3.7	—	11	—	—	—	—
41	Rh	17	1.9	—	—	2.2	—	13.7	13.2	9.8	—	3	—	—	—	—
44	A-O	—	—	—	—	—	10.6	12.9	22.3	—	35.6	96	—	—	—	—
17	B-O	—	—	—	—	33.0	—	—	35.0	34.0	26.0	60	—	—	—	—
18	Rh	—	—	—	—	—	—	—	20.0	10.3	10.1	60	—	—	—	—
19	A-O	—	—	—	—	14.9	—	17.3	18.3	22.0	15.8	78	—	—	—	—
24	Rh	—	6.8	expired during exchange ½ hr. after birth												
42	Rh	4.0	1.2	expired during exchange ½ hr. after birth												

An analysis of the correlation between the clinical status and the bilirubin determination in 31 patients treated during 1955 and 1956 may be drawn from table I; this table is arranged according to the number of exchange transfusions required for each infant during the neonatal period. In table II the same data are given for the 15 infants who did not require treatment. In this series

TABLE II

Hemoglobin Levels at Birth and Serial Bilirubin Determinations in 15 Infants Mildly Affected with Hemolytic Disease of the Newborn Who Did Not Require a Transfusion

Case No.	Incompatibility	Hg., Gm.	Cord Blood, Mg./100 Cc.	Serum Bilirubin Levels (Mg./100 Cc.), Hours After Birth									Treatment
				6	12	18	24	36	48	60	72	84	
10	Rh	15.5	2.9	—	–	4.3	—	—	9.0	—	15.0	12.0	none
13	Rh	17.0	2.0	—	–	—	5.5	—	7.4	14.6	15.5	—	none
14	Rh	17.0	1.9	—	–	—	—	—	—	—	14.3	15.1	none
32	Rh	18.0	3.2	—	–	11.0	—	—	13.7	—	20.3	17.0	none
35	Rh	18.5	2.8	—	–	—	5.6	—	3.0	—	—	—	none
36	Rh	17.5	2.8	—	–	—	5.4	12.7	13.9	10.5	—	—	none
20	B-O	—	—	—	–	—	—	13.6	—	19.8	23.8	14.3	none
21	Rh	20.0	—	—	–	8.8	12.0	—	16.6	—	—	12.9	none
22	Rh	—	—	—	–	—	7.3	—	13.5	9.0	10.1	—	none
39	Rh	20.0	2.6	6.0	–	—	7.2	—	—	—	5.0	—	none
46	A-O	—	—	—	–	—	—	10.9	13.5	19.3	—	—	none
47	Rh	17.0	—	—	–	—	—	9.0	10.0	13.0	10.0	—	none
48	Rh	20.0	—	6.6	–	—	—	—	—	—	—	—	none
30	Rh	18.5	3.8	—	–	—	—	—	14.4	21.0	27.0	—	none
49	A-O	20.0	—	6.0	–	—	—	—	—	—	—	—	none

TABLE III
Relationship Between the Hemoglobin and Bilirubin Levels at Birth in 30 Affected Infants

Group	No. of Cases	Hg. (Gm.) from Heel Blood		Bilirubin (Mg.) in Cord Serum	
		Average	Range	Average	Range
Hg. less than 15 Gm. at birth	13	12.4	11-14.7	6.03	2.1-12
Hg. greater than 15 Gm. at birth	17	17.2	15-20	3.37	1.9-4.5

of 46 cases, 13 infants required more than one exchange, 18 infants required one exchange, and 15 infants did not require transfusion therapy. All but 2 hydropic infants who expired within the first hour of life have survived and are now being followed in the outpatient section of our pediatric department.

In our clinical program we have not relied on the bilirubin levels in cord blood to determine the need for exchange transfusion at birth; however, these data were collected for the purpose of evaluating the relationship of the degree of intrauterine hemolysis in the clinical course. The correlation between levels of hemoglobin and bilirubin in cord blood at birth is shown in table III. It is seen that, in general, low hemoglobin levels obtained from heel blood at birth correlated with the higher levels of cord serum bilirubin. The infants with an average hemoglobin level of 12.4 Gm. at birth (ranging from 11 to 14.7 Gm.) showed a cord serum bilirubin average of 6.0 mg. per 100 cc., with a range from 1.2 to 12 mg. per 100 cc. In no infant whose hemoglobin level in cord blood was greater than 15 Gm. did the bilirubin level exceed 4.5 mg. at birth. This correlation is a measure of the degree of hemolysis in utero and, therefore, of the severity of the disease; however, more assistance is obtained regarding the decisions for an exchange transfusion immediately after birth from the low hemoglobin values than from the bilirubin levels in cord blood. For example, in cases 7, 8, and 40 (table I) exchange transfusion was indicated shortly after birth because of hemoglobin values of 11 Gm., and in these cases the bilirubin levels in cord blood were below 5 mg. per 100 cc. in each case. In 13 of the 14 infants who were transfused in the first 12 hours of life, the hemoglobin value at birth was less than 15 Gm. The bilirubin and cord blood level in these infants was above 5 mg. per 100 cc. in 57 per cent of these cases and below 5 mg. per 100 cc. in 43 per cent of the cases.

Although high levels of serum bilirubin in cord blood, that is, above 5 mg. per 100 cc., are shown by these data to correlate with the severe degree of intrauterine hemolysis, it is true that levels below 5 mg. per 100 cc. in the cord serum do not predict that an exchange transfusion will not be required during the newborn period.

Unless the severity of the disease at birth is such that an exchange transfusion is required shortly after birth, the rate of elevation of the bilirubin levels is the most reliable single criterion of the degree of hemolysis in the first few hours of life. The fall in the hemoglobin levels after birth may be deceptively slight as a result of the active erythropoiesis and delivery of new cells that compensate for the degree of blood destruction. The position of the hemoglobin values and the bilirubin levels shifts in terms of clinical importance after the birth of the infant.

In infants exhibiting a rapid rise in the bilirubin level within the first 24 hours after birth, it can be anticipated that a critical level will be reached within the next few hours. In figure 1 a comparison is given between the rise in the serum bilirubin levels recorded at intervals after birth in a group of infants who required exchange transfusions and in a group of infants whose degree of illness was mild and who did not require the trans-

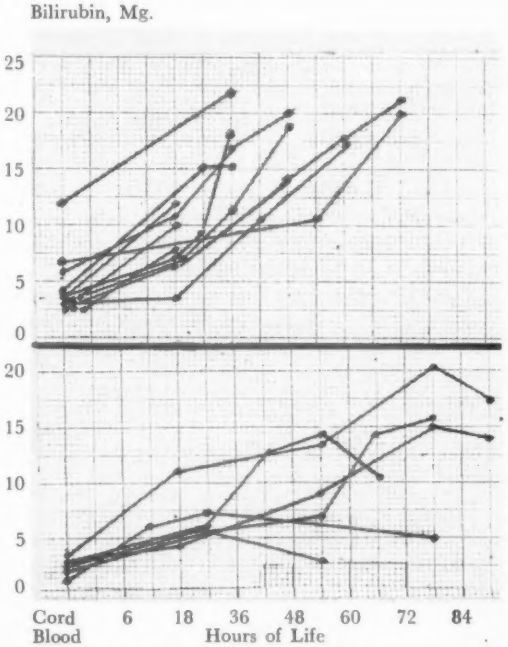

Fig. 1. Rate of increase in bilirubin levels. (Top) Before transfusion, in a group of affected infants who required exchange transfusion. (Bottom) In a group of mildly affected infants not requiring transfusion.

fusion. It is seen that the rate of increase in the bilirubin levels was more rapid in the babies who received exchange transfusion than in the group not receiving it. The rapid rise occurred within the first 24 hours of life in the infants who received exchange transfusions. The maximum elevation was reached in the group not receiving it only on the third day of life. A "critical" level of bilirubin that would serve as a guide for the exchange transfusion must vary necessarily with the age of the infant. A single determination within the first 24 hours of life has very little or no value. The serial determination at intervals of 6 to 12 hours will establish a curve that enables the clinician to obtain the maximum value of the test. A steeply rising level of bilirubin within the first 24 hours of life to a maximum of 12 to 15 per cent anticipates a further rise within the next few hours and justifies an exchange transfusion at this point. When the infant is somewhat older, 48 to 72 hours, it is still true that the rapid increase in the level is ominous, but there is, perhaps, less urgency to proceed with the exchange transfusion until the level of 20 mg. per 100 cc. or above is exceeded.

In determining the need for repeated exchange transfusions the same principle holds; for example, in case 6 (table 1) the bilirubin level in cord blood was 3.8 mg. per 100 cc. and the hemoglobin level in cord blood was 13.2 Gm.; at age 6 hours, the bilirubin level was 12 mg. per 100 cc., and an exchange transfusion was given, after which the level fell to 7.8 mg. per 100 cc.; and at age 36 hours, when the bilirubin level had risen to 26 mg. per 100 cc., another exchange transfusion was given and the clinical course was satisfactory thereafter. In case 37 (table I) the bilirubin level in cord blood was 2.6 mg. and the hemoglobin 16 Gm.; at 18 hours the bilirubin level was 9.8 mg. per 100 cc. and the first exchange transfusion was given; at age 36 hours the bilirubin level had risen to 19 mg. per 100 cc. and a second exchange was given, but the level failed to fall; and at 72 hours the third exchange transfusion was performed, after which the level fell to 12.1 mg. per 100 cc. within 6 hours. The clinical course has remained entirely normal to date.

Clinical signs of encephalopathy developed in only 1 of the 46 infants in this series of cases. Case 23 was the third affected child in a family of 5 siblings, 2 of whom had severe erythroblastosis. The infant was moderately ill at birth, exhibiting pallor and enlargement of the liver and spleen, but no cardiac or respiratory difficulty; hemoglobin level in the heel blood was 12 Gm. An exchange transfusion was performed immediately after birth. The cord serum bilirubin level was obtained later; it was 12 mg. per 100 cc. Before the completion of the transfusion, icterus of the skin was noticeable. Fourteen hours after the first exchange transfusion the infant was deeply jaundiced and the serum bilirubin level was 22.3 mg. per 100 cc.; a second exchange transfusion was done. At age 24 hours the bilirubin level had fallen to 14 mg. per 100 cc.; at age 35 hours, the clinical signs were suggestive of a mild encephalopathy, that is, lethargy, a high-pitched cry, a weak Moro response, and poor sucking efforts. Transfusions were repeated at age 36, 61, and 87 hours for bilirubin levels of 28, 38, and 45 mg. per 100 cc., respectively, at these times. The infant remained lethargic during this period, but no further signs of encephalopathy developed. After the third day of life the jaundice gradually receded; it cleared up by the fourteenth day of life. The baby is now 15 months of age, and there are no physical or neurological signs of the sequelae of kernicterus.

The early signs of kernicterus are: apathy or excessive drowsiness, recurrent vomiting, oculogyric movements of the eyes, a defective Moro reflex (either exaggerated or weak), and tremors of the extremities. The late signs are opisthotonos and athetoid movements of the hands and spasticity of the extremities.

When the early signs of encephalopathy disappear after exchange transfusion therapy there is still a risk that irreversible damage has occurred within the central nervous system, but, when neurological symptoms do not occur during the neonatal period, the prognosis is good regarding freedom from the late sequelae of kernicterus. In a follow-up study of 100 affected children, aged 1 through 5 years, who had recovered from hemolytic disease of the newborn infant, Jones[17] found that, of the cases with definite signs of encephalopathy in the newborn period, all showed neurological evidence of one or more of the late manifestations of kernicterus, such as varying degrees of athetoid spasticity, defects in mental development, or hearing loss. Of the infants who showed only transient and mild symptoms of encephalopathy in the newborn period, 33 per cent showed late neurological effects. Of the children who did not show symptoms of encephalopathy in the newborn period, only 4 per cent showed even minimal signs of the late sequelae. If the late manifestations of the neurological damage were absent, Day[18] found that the prognosis for normal mental development was good. It is the prevention of the serious and incapacitating sequelae of the neurological complications that argues most strongly

for the careful application of exchange transfusion therapy in this disease.

Summary

1. A short discussion of the mechanism of hyperbilirubinemia in the newborn period has been given.

2. Data have been presented on the serum bilirubin levels obtained from cord blood serum and from heel blood samples obtained at intervals throughout the neonatal period in a series of 46 infants with hemolytic disease of the newborn, 31 of whom were treated with exchange transfusions in 1955 and 1956.

3. The value of serum bilirubin determinations as a guide to the use of exchange transfusions has been discussed.

4. Clinical assessment of the degree of the severity of the disease at birth and the degree of anemia are more reliable than bilirubin levels in cord blood for determining the need for prompt use of the exchange transfusion shortly after birth.

5. Serial bilirubin determinations that permit an evaluation of the rapidity of the progressive hyperbilirubinemia provide reliable criteria for timing the need for the first exchange transfusion and for repeating it in treatment of hemolytic disease of the newborn infant.

6. No death occurred from kernicterus in this series of 46 cases in which an attempt was made to hold the bilirubin levels under 20 mg. per 100 cc. In only 2 cases did the level exceed 25 mg. per 100 cc.

7. Transient signs of mild encephalopathy developed in 1 patient in whom the hemolytic process was severe enough to require that she have five exchange transfusions.

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Ruth Renter Darrow and the Etiology of Hemolytic Disease of the Newborn Infant

Heyworth N. Sanford, M.D.

DIAMOND, BLACKFAN, AND BATY in 1932 successfully correlated three unrelated conditions occurring in the newborn infant—universal edema of the fetus, anemia of the newborn infant, and icterus gravis neonatorum—into one clinical entity, which was designated as erythroblastosis neonatorum, and which is now called hemolytic disease of the newborn infant. However, the etiology of these three apparently unrelated conditions was entirely obscure and was the cause of much speculation.

In 1938 there appeared in the *Archives of Pathology* an article entitled "Icterus Gravis (Erythroblastosis) Neonatorum" by Ruth Renter Darrow. As was usual in her studies, it was a very thorough description of this condition and correlated many points of the clinical and pathological picture. It also contained a full discussion of the various theories that were in vogue at this time. As a matter of fact, there were 10 very plausible explanations given for this condition, each of which was accepted as the true explanations by disciples of the masters who propounded them. Unfortunately, this made rather arduous reading, and, as the article ran about 40 pages, the reader was very liable to miss an explanation that Dr. Darrow herself proposed as a theory for the etiology of this condition. Furthermore, since this journal was not one that was read by the average practicing pediatrician, very little attention was paid to it. Here is the essence of her observations.

> "Two pathological processes are postulated as responsible, to varying degrees, for the symptoms and changes in the affected child. These are: (1) abnormal destruction of erythrocytes, and (2) injury of the liver. . . . If the possible mechanisms giving rise to destruction of erythrocytes are reviewed, it is found that all may be eliminated from consideration save one, the destruction of red cells by some form of immune reaction . . . The mother is actively immunized against fetal red cells or some component of them. The immunization may conceivably occur as the result of an accident within the placenta whereby the fetal cells or their hemoglobin gain entrance to the maternal blood sinuses. The antibodies formed in the maternal organism may then pass to the child, the placenta, or possibly to a greater extent through the colostrum and milk since the diminution of red cells in congenital anemia appears to be most acute following birth . . . Such a transfer of immune bodies from an actively immunized mother to the fetus or newborn child sets up in the offspring a state of passive immunity. Such an immunity is relatively short lived and would eventually disappear completely, leaving no trace to be passed on to a succeeding generation. Furthermore, each child born and suckled subsequently to this active immunization in the mother would possess to a greater or less degree a passive immunity to the specific antigen, while any children born before the immunization of the mother would be entirely unaffected."

Three years later (in 1941) Levine, Katzin, and Burnham reported that the Rh factor found in the blood of certain individuals by Landsteiner and Wiener was the cause of this condition. The mode of action of these formed antibodies has been found to be exactly the same as Dr. Darrow's postulated explanation.

Some of us, unfortunately only a few, have been given the ability to formulate in a concrete manner a causative explanation from the known established facts. All of these facts are apparent for each and everyone of us to see, if we have the ability and persistence to interpret their action. Dr. Darrow, with no more evidence and no more established facts then were available to all of us, was quite able to postulate in a logical manner the correct explanation of this condition as satisfactorily as if all of the later findings had been given to her.

It is for this unique ability to reason well that we will all miss Ruth Renter Darrow and her faculty of placing apparently unrelated facts in a logical sequence that helped to change a confused picture to a clear and sensible one.

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Review of Thrombocytopenic Purpura in Relation to Pregnancy and the Newborn Infant

Bertha L. Isaacs, M.D.

PURPURA DURING PREGNANCY has been reported often since the original account by Barnes in 1867, but its etiology was not known until discovery of platelets was made by Bizzozero. Many more years were to elapse before the pathogenesis of idiopathic thrombocytopenic purpura was understood; however, during the intervening years many theories were advanced.

Neonatal purpura has been reported less than a hundred times in the literature, and many of these reports lack adequate clinical and hematological data. Because mortality rates for the pregnant woman with thrombocytopenic purpura and for her infant are high, the need for prompt diagnosis and alert management throughout the puerperium and neonatal periods is apparent.

Development of knowledge about platelets was slow after Bizzozero recognized them as specific structures in the mesenteric vessels of rabbits and guinea pigs and demonstrated their adhesive quality in relation to thrombus formation and coagulation of blood. Nineteen years elapsed before J. H. Wright showed that platelets originated from the bone marrow of the adult. Many investigators have attempted to determine whether platelets are living structures with metabolic activities or merely fragments of cytoplasm, but current opinion appears to attest to their viability. The study of platelets has been hampered by their small size, fragility, short span of life, and the technical difficulties encountered in preparing pure suspensions. The five days required for the formation, maturation, and delivery of platelets to the circulating blood has a useful clinical connotation when effort is being made to evaluate the response to treatment in a patient with acute severe idiopathic thrombocytopenic purpura. The following tabulation summarizes the significant facts in regard to platelets:

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PLATELET DATA

1. Origin is from megakaryocytes of bone marrow.
2. Metabolic investigations indicate living structure.
3. Normal life span is from three to five days.
4. Normal average count in peripheral blood is 250,000 to 500,000 per cubic millimeter.
5. Platelet types have been identified; they are not related to red blood cell types.
6. Platelets are antigenic.
7. The platelet count in the peripheral blood of newborn infants averages from 100,000 to 150,000 per cubic millimeter and increases slowly to a normal adult level by the third month.
8. Premature infants average lower platelet values and the return to normal is slower.
9. In the pregnant state, circulating platelets often rise before the onset of labor, decrease during labor, and return to normal in 24 hours.
10. A decreased platelet count has been reported in toxemia of pregnancy.

Nearly all the theories advanced to explain the etiology of idiopathic thrombocytopenic purpura recognized the importance of the spleen. At various times the thrombocytopenia has been attributed to any of the following: (1) deceased rate of platelet formation from the megakaryocytes; (2) increased rate of platelet destruction by the spleen; and (3) change to both megakaryocyte and circulating platelets.

Frank believed that megakaryocytic formation was inhibited by a splenic humoral factor. Transient thrombocytopenia was demonstrated by Troland a Lee when extracts of spleen from thrombocytopenic

patients were injected into laboratory animals. Pohle and Meyer, and Tocantins, failed to confirm this work, but reaffirmation was given by Moolten, Limarzi, and Schleicher, and Dameshek and Miller.

Other investigators thought that the spleen caused an increase in the rate of destruction of platelets by means of circulating lysins or agglutinins or by selective sequestration and phagocytosis of platelets already sensitized by a circulating factor. Upon the latter premise Knazelson, in 1916, first advised splenectomy as the treatment of choice for idiopathic thrombocytopenic purpura. The beneficial effects of splenectomy were reviewed by Wiseman and co-workers, and the high remission rate following surgical removal of the spleen has established this as the treatment for chronic idiopathic thrombocytopenic purpura.

The first clinical support to Frank's theory that a humoral factor was elaborated by the spleen was given by the evidence that infants born of mothers with chronic idiopathic thrombocytopenic purpura were frequently purpuric. This fact also suggested that transplacental transmission of the thrombocytopenic factor occurred.

The second clinical observation in support of a humoral factor, immunological in nature, came from Evans' recognition that patients with idiopathic acquired hemolytic anemia often had episodes of idiopathic thrombocytopenia. In an earlier report he revealed that a constant demonstrable antibody for erythrocytes was present in acquired hemolytic anemia. Therefore, it seemed natural to postulate that an analagous autoantibody existed for platelets and that this could be responsible for thrombocytopenia.

Still greater evidence in support of the existence of a humoral thrombocytopenic factor of immunological nature was afforded by the experimental results of Ledingham, Bedson, Tocantins, and others. They demonstrated thrombocytopenia in animals injected with heterologous antiplatelet sera and also produced purpura if the animals' capillary endothelium was damaged by prior injection with anti-red-blood-cell sera. Splenectomy reduced this sensitivity if performed before injection with the sera.

Grandjean's demonstration in 1948 that in vitro addition of quinine to the blood of a patient who had recovered from thrombocytopenic purpura induced by the drug caused lysis of the patient's platelets was another sign that an immunological factor was operative in the pathogenesis of thrombocytopenia. Recently, Hoffmeister, Gorthey, and Olinger reported the case of a woman who became nauseated and vomited after a dose of a quinine preparation. Four years later she developed thrombocytopenic purpura after a small dose of quinine was given for induction of labor. Particularly interesting was the fact that her serum still contained antibodies against quinine nine months later and that her serum agglutinated, lysed, and prevented the clotting of her blood as well as that of a normal person's blood. This was clear-cut evidence of passive transfer of an antibody.

Other agents such as quinidine, allyl-isopropyl-acetyl-carbamide (Sedormid), analgesics, antihistaminics, certain sedatives, antibiotics, estrogens, foods, and cosmetics also have been known to produce severe temporary thrombocytopenia within a few hours in sensitized persons by immunological processes that do not actually destroy the megakaryocytes. This type of induced thrombocytopenia is usually self-limited, brief, and disappears upon withdrawal of the offending agent.

Identification of the antibody in thrombocytopenic purpura as a platelet agglutinin was soon made. Several different groups of investigators detected platelet agglutinins in the sera of some patients; others produced thrombocytopenia in normal persons by transfusions of plasma from patients with idiopathic thrombocytopenic purpura. It was also shown that normal platelets transfused into patients with idiopathic thrombocytopenic purpura had less than a normal life span.

Another type of antigen-antibody reaction was predicted by Ruth Renter Darrow when she reviewed the literature on erythroblastosis fetalis in 1941. Her predication preceded by only a few months Levine's identification of the Rh factor as an antigenic substance responsible for the production of isoagglutinins in the mother with the formation of an antibody capable of crossing the placental barrier and producing hemolytic disease of the newborn infant. The occasional appearance of thrombocytopenia in this condition is of interest because of the indication that separate isoimmunization to red blood cells and platelets may coexist. The existence of ABO groups and types in platelets has been demonstrated but their significance is not clear.

There is one report of fetal-maternal platelet incompatibility in which the mother's platelet type differed from that of the father's and child's, and another report was made of maternal isoimmunization to fetal platelets with development of thrombocytopenic purpura in only one of fraternal twins.

Wiener and Hedenstedt and Naeslund have shown that the erythrocytes of the fetus do not enter the maternal circulation unless the placenta

is abnormal, but recent studies show that fetal red blood cells do enter the maternal circulation in many normal pregnancies as well as in erythroblastosis fetalis.

The studies of Creger and Steele indicate that 5 cc. of fetal blood are present in the maternal circulation. Because this is greater than the minimal amount known to be antigenic when injected intravenously, it supports Levine's suggestion that the low incidence of erythroblastosis fetalis in relation to the frequent combination of an Rh-negative mother with an Rh-positive fetus is due to low antibody-forming ability of some mothers.

Thrombocytopenia without evidence of the clinical syndrome of acute idiopathic thrombocytopenic purpura has been shown to occur in toxemias of pregnancy. Platelet counts below 100,000 per cubic millimeter are unusual in normal pregnancy or delivery, but reduction to zero has been reported by Ferguson in 1 pre-eclamptic and 2 eclamptic patients in labor. No hemorrhagic events were associated with the thrombocytopenia nor was there any apparent relation between convulsive episodes and platelet disappearance.

The subject of neonatal thrombocytopenic purpura has been covered so adequately by several reviewers that only a brief discussion of pertinent factors is given in this paper.

The pathogenesis of this syndrome has been recognized to be the transplacental passage of either autoagglutinins or isoagglutinins for platelets. Mothers with idiopathic thrombocytopenic purpura may give birth to infants with thrombocytopenic purpura. In fact, half of the infants are purpuric if the mother's platelet count is normal, but three-quarters are purpuric if the mother's platelet count is low at the time of delivery.

Reports from the literature (see table I) indicate that fetal and maternal mortaliy figures may reach 15.1 per cent and 2.1 per cent, respectively,

TABLE I.
Recent Reports of Neonatal Thrombocytopenic Purpura

Authors	Case Reports	Summary of Case Reports
Peterson, Jr., and Larson (1954)	1	1. Authors reviewed 65 cases of infants in literature (born of 47 mothers) in which maternal mortality was 2.1% and fetal mortality was 15.1%. 2. Of these, the infant mortality was 11.1% when splenectomy was done before pregnancy; 30.7% when splenectomy was done after pregnancy; and 11.5% when no splenectomy was performed before or during pregnancy. 3. Purpura appeared in 80.4% of infants whether or not splenectomy was performed before delivery. 4. If the platelet count of the mother was normal at delivery, 45.5% of the infants were purpuric. If the platelet count of the mother was low at delivery, 74.4% of the infants were purpuric.
Chalmers (1954)	1	1. No report was made of infants' platelet counts. All apparently survived.
Vandenbroucke and Verstraete (1955)	1	1. Infant's platelet count at birth was 10,000 per cu. mm. 2. Mother's platelet count at delivery was 46,000 per cu. mm. 3. There was no neonatal purpura in her 3 children previously born. 4. Antiplatelet agglutinins were present in the serum of the mother and her infant (persistent in mother but disappeared in infant).
Schoen, King, and Duane (1956)	1	1. Mother's diagnosis was idiopathic thrombocytopenic purpura 2½ years before this admission and splenectomy. 2. Infant had petechiae over the entire body 15 minutes post partum which lasted for 3 weeks. 3. Infant's platelet count was 6,500 per cu. mm. (8 hours post partum). 4. Infant's platelet count was normal when infant was 2 months old. Cortisone treatment was given for the first 24 days of life. 5. Isoagglutinins for the baby's platelets were found in the mother. 6. The mother's serum agglutinated platelets of several normal patients tested.
Kaye and Dufalt, Jr. (1957)	1	1. Mother's platelet count was 49,250 per cu. mm. at seventh month of pregnancy. 2. Fluctuating thrombocytopenia and menomenorrhagia were present for 3 years prior to this pregnancy. 3. She had a normal delivery 4 years before. 4. Cortisone was administered 10 days before delivery. Platelet counts of mother and baby were normal during and after delivery. Circumcision at 1 month was uneventful.

in thrombocytopenic purpura during pregnancy. In smaller series these figures are even greater. The fetal death rate is three times higher if splenectomy has to be performed during gestation than it is if no splenectomy is done before or during gestation.

Idiopathic thrombocytopenic purpura may become manifest before or during pregnancy. If the available evidence indicates that the thrombocytopenia is of the autoimmune variety, but not associated with drug or food sensitivity, splenectomy provides the most effective treatment before or during the early months of pregnancy. Adrenocortical hormones may be used to manage some patients if there are contraindications to surgery or if gestation has reached the third trimester. Although the mechanism by which these hormones effect control is not proved, it is known that their use preserves the integrity of capillary endothelium, thereby reducing the bleeding tendency. Possibly these hormones also interrupt the antigen-antibody reaction.

Clinical experience indicates that routine administration of corticosteroids during the last two weeks of gestation appears to reduce the risk or severity of thrombocytopenic purpura in the newborn infant, irrespective of previous splenectomy in the mother. Because 75 per cent or more of the infants born of mothers with this condition survive, and because the purpura and thrombocytopenia disappear spontaneously during the first one to four weeks of life, the value of administering adrenocortical hormones after birth is open to question.

Transfusion of platelets is not advised because of the risk of immunizing the infant to new platelet antigens, nor should exchange blood transfusions be done unless there is concomitant hemolytic disease of the newborn infant.

Delivery should be effected in the manner consistant with the best obstetric practice, with due consideration given to avoidance of quinine induction of labor, to birth trauma attendant upon forceps delivery, or to excessive manipulation of the infant during the first 24 hours of life. No undue complications of cesarean section, bleeding from episiotomy, or post-partum hemorrhage need be anticipated if the mother's thrombocytopenic purpura has been adequately controlled. On the other side, elective surgery (a case report showing this type of reaction is to be published) should be withheld during the first year of life because of the chance that platelet antibodies have persisted and might cause reappearance of thrombocytopenic purpura.

Breast feeding of infants born under these conditions is contraindicated.

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†Contains an extensive bibliography to which referral may readily be made.

CASE REPORT

A Case of Severe Erythroblastosis Fetalis (Fetal Hydrops) with Recovery

Clementine Frankowski, M.D.

THIS IS A CASE report of an infant with fetal hydrops who, at the time this report was written (when the infant was 8 months old), appeared normal mentally and physically except for slight weakness. Rh-positive, type O, blood (the father's) was given first, according to Dr. Darrow's concept of the desensitizing action, as well as the oxygen-carrying action, of Rh-positive blood cells.

The mother of this infant, Mrs. V. R., aged 37, gravida VII, Para IV, had one child (her third) who was affected with erythroblastosis. A small transfusion had been given, but the infant died 16 hours after delivery. The fourth child was not affected; it was born in January, 1955, and was Rh negative.

CASE REPORT

The patient's antibody titer was 1:64 at the time the fifth baby was born. She had taken vitamins, calcium, and 8 mg. twice daily of chlorpheniramine maleate prenatally and seemed to progress normally. She started labor six weeks prematurely and was delivered spontaneously after six hours and 21 minutes of a living female child who weighed 6 pounds and 4 ounces.

At birth the child was cyanotic, with grunting and labored respiration; her legs were edematous, the abdomen was distended, the liver and spleen were enlarged, and numerous rales were heard over both lungs. The placenta was large and edematous, as was the umbilical cord. The amniotic fluid was very dark green, and the cord blood showed the following: hemoglobin, 7.2 Gm. per 100 cc. (45 per cent); erythrocytes, 2.46 million per cubic millimeter; and leukocytes, 19,000 per cubic millimeter, with a differential of 60 polymorphonuclear leukocytes (10 stabs, 50 segmented), 1 basophil, 3 eosinophils, 33 lymphocytes, 3 monocytes, and 260 nucleated erythrocytes per cubic millimeter (table I). The Coombs' test was positive, and the serum bilirubin level was 2 mg. per 100 cc. The infant and her father showed Rh-positive, type O, blood, while the mother had Rh-negative, type O, blood.

The baby was placed in an Armstrong crib, received oxygen, and was given vitamin K. About an hour and a half after birth she was given 100 cc. of Rh-positive blood (the father's) followed by 2.5 cc. of calcium gluconate. The baby appeared clinically better after receiving the blood. Seven hours post partum, the hemoglobin level rose to 12.7 Gm. (79 per cent) per 100 cc., the erythrocyte count was 4,150,000 per cubic millimeter, the serum bilirubin level was 12.2 mg. per 100 cc., and there were 170 nucleated erythrocytes per cubic millimeter. Eight hours after delivery an exchange transfusion was given. Four hundred cubic centimeters of the father's blood were injected and 350 cc. were withdrawn. Two and a half cubic centimeters of calcium gluconate were given after each 100 cc. of blood. On the next day the infant appeared more jaundiced and cyanotic, respiration was more irregular, and the abdomen was more distended. The serum bilirubin level went up to 13.5 mg. per 100 cc. The hemoglobin level and erythrocyte count went down. During that evening the serum bilirubin level rose to 15.5 mg. per 100 cc. One hundred cubic centimeters of Rh-negative, type O, blood were given from the blood bank. On the third day the baby's condition was critical: cyanosis was marked, the abdomen was markedly distended, the legs and arms were edematous, and the temperature was 104.4 F. The serum bilirubin level was 22 mg. per 100 cc., the hemoglobin level and erythrocyte count went up, and there were 207 nucleated red blood cells per cubic millimeter. Another exchange transfusion was given of 362 cc. of Rh-negative, type O, blood, and 288 cc. were removed. The transfusion was stopped at this point because cyanosis increased and respiration became very shallow. The infant was given 0.5 cc. of diphenhydramine intramuscularly and put in isolet. At 3 p.m. the following afternoon, the temperature fell to 101 F., and the skin color looked better; however, respirations were still irregular. On the fourth day, the temperature was 99 F., cyanosis was still present, jaundice was decreased, and the abdomen was still markedly distended. The serum

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TABLE I
Laboratory Work in Hospital

Date, 1956	Time	Bilirubin, Mg./100 Cc.	Hg., Gm./100 Cc. (%)	Color Index	RBC/Cu. Mm.	WBC/Cu. Mm.	Differentials						
							Polys.		Eosinopils	Basophils	Lymphocytes	Monocytes	Nucleated RBC/Cu. Mm.
							Seg.	Stab					
5/10	10:21*	2.0	7.2 (45)	—	2,460,000	19,000	50	10	3	4	33	4	260
5/10	5:00†	12.2	12.7 (79)	.96	4,150,000	35,900	38	26	–	–	36	—	170
5/11	9:00*	13.5	10.5 (65)	—	3,480,000	30,000	44	45	–	–	5 (6 juvenile)	—	125
5/12	4:00†	15.5	—	—	—	—	—	—	–	–	—	—	—
5/12	9:00*	20.0	12.7 (79)	—	4,100,000	—	38	30	2	–	3	3	207
5/12	5:00*	22.0	—	—	—	—	—	—	–	–	—	—	—
5/13	—	15.5	—	—	—	—	—	—	–	–	—	—	—
5/14	9:00*	12.2	— (90)	—	—	—	—	—	–	–	—	—	—
5/15	—	15.5	15.7 (98)	—	4,850,000	18,700	65	18	2	–	18	2	12
5/16	9:00*	—	16.0 (101)	—	5,270,000	12,900	48	10	3	–	33	5	—
5/21	9:00*	6.0	14.6 (98)	—	4,650,000	12,800	56	9	2	–	26	7	—
5/26	9:00*	—	14.0 (87)	1.00	4,385,000	8,400	65	9	2	–	20	14	—

Hg.=hemoglobin. Polys.=polymorphonuclear leukocytes. Seg.=segmented.
*=a.m.
†=p.m.

bilirubin level was 15.5 mg. per 100 cc. One hundred centimeters of 5 per cent glucose were given through the umbilical vein. On the fifth day the skin color looked better, there were petechiae on the skin, and the baby vomited some blood. The infant took fluid orally on the fifth day for the first time, and 100 cc. of 5 per cent glucose were injected into the umbilical vein. On the sixth day the serum bilirubin level went down to 12.2 mg. per 100 cc. The baby received 300,000 units of penicillin twice daily for the first 10 days and glucose subcutaneously at least once daily. The infant's condition remained the same until the tenth day, and on the eleventh day oxygen therapy was stopped.

The baby gradually improved but remained quite flaccid and would become a little cyanotic after feedings. She remained in the hospital until the sixteenth day. When she left the hospital she weighed 5 pounds and 6 ounces; the hemoglobin level was 14 Gm. per 100 cc. (87 per cent).

At present, the baby is 8 months old, weighs 15.4 pounds (table II), is alert, and is developing normally.

TABLE II
Weight* Record and Blood Counts Since Leaving Hospital

Date	Weight Lb. and Oz.	Hg., Gm./100 Cc. (%)	Color Index	RBC/Cu. Mm.	WBC/Cu. Mm.	Differentials				
						Polys.		Eosinopils	Monocytes	Lymphocytes
						Seg.	Stab.			
6/1	6 and 5½	12.1 (63)	—	3,830,000	10,600	32	2	5	–	60
6/11	6 and 5	8.1 (51)	.79	3,260,000	7,100	—	–	–	–	—
6/19	6 and 13½	8.4 (53)	.85	3,180,000	7,200	20	2	2	–	76
6/26	7 and 5½	8.1 (51)	.83	3,000,000	8,000	26	6	4	–	63
7/3	8 and ½	8.1 (51)	.89	2,960,000	7,900	34	2	1	–	63
7/10	8 and 12	9.2 (57)	1.00	2,860,000	4,600	13	4	1	2	72
7/17	9 and 7½	9.8 (—)	.98	3,100,000	9,000	35	5	–	–	60
8/13	11 and 2½	11.5 (72)	.96	3,840,000	6,700	22	2	–	1	75
9/18	12 and 4	11.6 (73)	.86	4,250,000	10,000	34	3	4	4	55
1/8/57	15 and 4	12.7 (—)	.96	3,140,000	7,500	17	1	–	4	78

Hg.=hemoglobin. Polys.=polymorphonuclear leukocytes. Seg.=segmented.
*Weight was 6 pounds, 4 ounces, during entire hospital stay

At the last examination the back muscles were still a little flaccid.

Comment

Recovery in this case is attributed to the use of the father's blood. The explanation of this treatment is based on the concept that anaphylactic sensitization plays a major role in the pathogenesis of erythroblastosis fetalis. The value of intravenous transfusion of type-specific erythrocytes containing the antigen is in its desensitizing effect.

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Dental Care for Bedfast Patients

A study designed to lead to improved dental care for the chronically ill and aged was announced in May by the Public Health Service. The study will be made by Montefiore Hospital and the Beth Abraham Nursing Home, both of New York City, in co-operation with the Service's Division of Dental Public Health.

The study concerns the development of portable dental equipment and methods for dental care of bedfast and homebound patients. Many such patients now get little or no such care because they cannot visit a dentist's office.

"It is important that information be collected on techniques and equipment for treatment of the bedfast and also for persons suffering from unusual conditions," Dr. Norman F. Gerrie, Chief of the Division of Dental Public Health, said. "The over-all effect of such service and the costs of providing it need to be evaluated. Technical and administrative problems also need to be ironed out."

The patients in Montefiore Hospital's Home Care Program and those of the Beth Abraham Nursing Home are representative of patients found in many institutions and among the homebound chronically ill.

A Public Health Service dentist and dental hygienist will accumulate data on dental needs of the homebound and institutionalized patients. Complete dental service will be provided to the approximately 400 in-patients in the Beth Abraham Home and to the approximately 80 patients in the Montefiore Hospital Home Care Program.

Medical Highlights

NEW STRAIN OF INFLUENZA VIRUS

Dr. Leroy E. Burney, Surgeon General of the Public Health Service, has established an advisory committee of physicians and health officers to consider precautionary steps in the United States against the current influenza epidemic in the Far East.

Epidemics in the Far East have been caused by a new strain of influenza virus which apparently is not controlled by current influenza vaccine. Much of the influenza caused by the new virus has been relatively mild, marked by a three or four day period of fever and other typical influenza symptoms.

No confirmed cases of the Far Eastern influenza strain in the United States have been reported to the Public Health Service.

The summer months normally are the months of lowest influenza incidence in this country. The disease usually is most prevalent in the fall and winter.

Dr. Burney said that the following precautionary measures have already been taken by the Public Health Service.

The Service has provided samples of the new influenza virus to manufacturers of vaccine in the U.S. Consultations with manufacturers will be continued during the coming weeks on the possibilities of producing vaccine for general distribution if this should be indicated. Influenza vaccine has been used successfully in the past, although it is sometimes ineffective against certain strains.

Foreign quarantine inspectors, which the Public Health Service stations at all international sea and air ports, are advising travelers from the Orient and the Philippines to see their private physicians if they develop a respiratory illness within 10 days after their arrival. The names and addresses of passengers who have a respiratory illness when they arrive in the United States are forwarded to the health officers of the communities to which they are going.

The Service has developed antigen testing agents for diagnosing infection caused by the new strain of influenza. These antigens will soon be sent to collaborating laboratories throughout the Western Hemisphere to help them determine whether cases of respiratory illness are caused by the new influenza strain. Until enough antigens are available to supply these laboratories, the Communicable Disease Center of the Public Health Service will make the identifying tests. All state health officers have been informed about the testing system.

State and local public health officials are being asked to encourage private physicians to report suspicious cases, so that there will be information on any cases of this type of influenza which might develop in the United States.

DRAMATIC DROP IN POLIOMYELITIS

Cases of all types of poliomyelitis in Canada in 1956 totaled 600, a decrease of nearly 59 per cent from the all-time high of 8,878 in 1953, according to a special report on poliomyelitis released by the Dominion Bureau of Statistics.

Only two provinces, Quebec and Ontario, showed slight increases in the number of cases in 1956 as compared with the preceding year. The national incidence rate of 3.7 per 100,000 population was the lowest recorded since 1945, when a rate of 3.2 was shown.

Paralytic cases decreased 67 per cent from 551 cases in 1955 to 369 in 1956, and were the fewest since 1950, when 284 cases were reported. The incidence rate at 2.3 per 100,000 population was also the lowest since 1950, when the rate was 2.1. New Brunswick, Quebec, and Ontario showed slightly higher incidence rates in 1956 than in 1955.

The proportion of cases in the age group 20 years and over was the highest recorded since 1949 (41.3) for the six provinces reporting age. This proportional increase, however, was due to the lower incidence at younger ages rather than to an increase in the age group 20 years and over. (From *Canadian Weekly Bulletin*, May 29, 1957.)

American Medical Women's Association

President's Message

When a cherished garment has been outgrown, it pinches and confines, hampering the freedom of action of the wearer and limiting his comfort and good function. Alterations can be made from time to time, but the day comes when the patches are not in harmony with the style and fabric. Sooner or later, a completely new garment must be made. The tailoring will follow the general pattern where it continues to be appropriate, but the new garment will reflect the individual's expectation for further growth with lines that are simple and standard enough to remain in style for an extended period of time.

The Constitution and Bylaws of AMWA resemble the much patched and altered garment. Some provisions that were of the utmost importance when the work of the Association was done entirely by member physicians are now cumbersome and antiquated. Revisions in some areas contradict the intent of another section. Our electoral procedure leaves much to be desired, and a number of other changes must be made if we are to have a democratic and vital organization.

In the fall of 1956 the branches were polled on the question of revision versus rewriting. The replies were unanimously in favor of complete rewriting rather than repair. Less than a week after the close of the Annual Meeting in June of 1957, a very carefully chosen, very dedicated committee with experience in AMWA and an understanding of the framework of other large organizations went to work on this very delicate and difficult undertaking. They have had a number of meetings and have spent many, many hours weighing each article and section, comparing the structure to that of other similar organizations and using reference material abundantly. No change has been introduced without ample discussion and a strong confidence that the final thought was the most appropriate for the present and future needs. Legal counsel will review it for sound wording and conformity with the laws for incorporated, nonprofit organizations in the state of New York before it is mailed to the membership for study, criticism, and approval.

If the work continues on schedule, the new Constitution and Bylaws will be in the hands of each member sometime in September. It is sincerely and earnestly requested that each person study it carefully and discuss it with other members and in branch meetings.

It is of the utmost importance for the Committee and for the welfare and satisfaction of the membership that all comments, criticisms, and suggestions be sent in as promptly as adequate deliberation permits. They should be in the hands of the Committee for evaluation well in advance of the Midvear Board Meeting to be held in Dallas on Nov. 14-17, 1957. The Committee will hold an open hearing for discussion of all controversial points. Branch delegates should be instructed by the group they represent to obtain clarification on any section or portion that has not met with approval.

Even a slight change in a constitution can precipitate a lot of heated spur-of-the-moment discussion on the floor, most of which is born of misunderstanding, and often generates more heat than light. It is hoped that this can be avoided at the time of the final vote in June of 1958 by giving the membership ample opportunity to express their opinions in the fall and to hear the Committee's reasons at the Midyear Meeting.

It is the wish of your President and the chairman and members of the Committee that the new Constitution be well tailored to the needs of the organization and fully acceptable to the membership. Only by your response can this be achieved in harmony and without undue delay.

Elizabeth S. Kahler

Association News

INTERNATIONAL

Booklet on Health Hints

The booklet "Health Hints for the Tropics" is recommended by the Department of Health, Education, and Welfare, U.S. Public Health Service, as a guide for health precautions to be observed generally by the international traveler. It may be purchased for 25 cents from the Editor of Tropical Medicine and Hygiene News, National Institutes of Health, Bethesda 14, Md.

Immunization Information for Foreign Travel

The following information may be helpful for members of AMWA who plan to attend the Congress of the MWIA in London next year, as well as for others who are traveling to foreign countries at any time.

The International Certificates of Vaccination or revaccination (against smallpox, yellow fever, and cholera) forms were revised in January, 1957. In the case of primary vaccination, the words "successful" and "unsuccessful" have been printed so that the vaccinator may check one or the other after inspecting the vaccination site. This has been done to assist quarantine officials of various nationalities and language backgrounds to interpret more readily and correctly the smallpox vaccination information; this should, in turn, facilitate activities at ports of entry.

Individuals may purchase the certificates from the Superintendent of Documents, Government Printing Office, Washington 25, D.C., for 5 cents a copy.

Smallpox and cholera vaccination certificates, to be valid, must be authenticated by a U.S. stamp approved for this purpose. The yellow fever vaccination certificate should bear the seal of the U.S. Public Health Service, and the certificate issued at other centers designated by the Public Health Service should bear the individual stamp assigned to the center. Presentation of a valid certificate by the traveler at ports of entry also helps to expedite quarantine activities.

International Co-operation

At the 1958 Annual Meeting of the AMWA the following resolution was adopted: "That the AMWA co-operate in the promotion of better international understanding and relations by studying the various governmental and nongovermental agencies working in this field."

The following report is presented for your information. If you would like additional information on any segment of work of the World Medical Association please write to the AMWA office.

Dr. Louis H. Bauer, Secretary General of the World Medical Association, reports, in a recent communication sent to the AMWA, on the worldwide activities of the W.M.A. and appeals for better understanding of the role this organization performs in creating, through the universal language of medicine, "the only road to peace"—understanding.

Dr. Bauer says: "W.M.A. has formulated an International Code of Medical Ethics and a modified Hippocratic Oath defining the ideals and obligations of physicians. W.M.A. has also set forth the basic principles that should protect the rights and status of doctors in all governmental social security schemes. And W.M.A. has done more than give lip service to these codes and principles. It is working day and night around the world to protect the profession wherever it is threatened by political interference that would prevent the doctor from serving his patients according to his scientific knowledge and his conscience. Recently, W.M.A. has defended the rights of the medical profession against attempts by various nonmedical organizations to draft a code of international medical law."

Other benefits derived from membership in the W.M.A. are solidarity and strength, by physicians coming together for the solution of their common problems; assistance to members of the U.S. Committee in making arrangements for foreign travel and in providing information on foreign medical meetings; introduction to medical leaders and teachers abroad; and promotion of international friendships and contacts of physicians. Membership in the U.S. Committee also brings the *World Medical Journal*, published every other month by

W.M.A., with world-wide information on medical progress and problems in the 53 countries whose national medical associations comprise the membership of W.M.A.

The W.M.A. speaks and works for your interests in its contacts with other world organizations concerned with health or medical care, such as, the World Health Organization of the United Nations, the International Labour Organization, the International Social Security Association, and the International Committee of the Red Cross.

Some other activities and accomplishments of W.M.A., of interest to every American physician are: (1) sponsorship of the First World Conference on Medical Education, at which DR. HULDA THELANDER of San Francisco represented the AMWA (the Second World Conference on Medical Education will be held in Chicago in 1959); (2) promotion of international exchanges of medical students and teachers and lectures and clinical teaching by traveling teams of physicians; (3) development of an international program to improve occupational health services; (4) promotion of a freer flow of proved therapeutic agents throughout the world by urging removal of unwarrantable trade restrictions and arbitrary licensing requirements in certain countries; (5) development of medical research, by promoting national pharmacopoeias and by defending the rights of individuals to name new drugs and agents they have discovered; (6) formulation of plans for a central repository of medical credentials, to enable qualified physicians of every country to file proof of their identity and qualifications with a safe and authoritative international source; (7) conducting of useful studies of many subjects of world-wide interest to physicians, such as, postgraduate medical education, hospital facilities, cult practices, medical advertising, and effects of social security legislation on medical practice; and (8) development of an international emblem for identification and protection of medical units and civilian physicians engaged in civil defense in wartime and promulgation of regulations stating the rights and duties of such physicians.

Dr. Bauer says: "The World Medical Association needs you—we invite you to add your name and your voice in guiding and strengthening this great organization."

The office of the World Medical Association is located at 10 Columbus Circle, New York 19.

LEGISLATION

"Patchwork" Chemical Laws

A conference of representatives from government, industry, and medicine is planned for this fall, to draft a model law, which will later be submitted to legislative bodies, regarding the labeling of hazardous chemicals. It is believed that a uniform law, which does not exist at present, is needed in this area.

According to the American Medical Association, "The proposed legislation is intended to reduce careless and ignorant handling of potentially harmful products in and around the home, in small businesses, and in other areas where control of overexposure is not as efficient as in the manufacturing process. The law will require informative labeling, listing of possible harmful ingredients, their potentialities for harm, directions for safe use, and first aid instructions."

There is great variance between state laws in their requirements for labeling and a number of regulatory acts at federal level, all of which confuse and lead to omissions of many of the necessary regulations.

Only 19 states conform to the Federal Food, Drug and Cosmetic Act of 1938, even though 40 per cent of all drugs sold are confined to interstate commerce.

In addition to the need for a uniform law, which is quite apparent, such legislation will offer greater protection to users of chemicals, will facilitate educating of the public to the significance of warning labels, and will obviate the need for special packaging and labeling for each state.

MEDICAL ECONOMIC NEWS

Cost for Maternal and Child Welfare

The Health Information Foundation has released an estimate of the amount spent by American families in 1956 for maternal and child welfare. More than 4 million births were recorded and about 950 million dollars were spent to pay the doctor, hospital, and other maternity bills. The average cost per family was $225.

Opportunities for Women in Medicine

AWARDS

Medical Education in Texas. The University of Texas Medical Branch in Galveston is the recipient of a large bequest from the late Dr. Samuel E. Thompson, to be used "to assist poor and deserving young men and women students, citizens of the state of Texas who without such assistance might not be able to obtain the advantage of a medical education." The loans will not exceed $3,000 per student and will be made at a rate of interest not to exceed 4 per cent.

Undergraduate. The Borden Company Foundation will again present scholastic awards in a number of medical schools to stimulate and reward research activity by undergraduates. The colleges select the recipients and administer the grants on a renewable five year basis.

Urology. Three prizes, $500, $300, and $200, are offered by the American Urological Association for the best essays on the result of some clinical or laboratory research in urology. Competition is limited to urologists who graduated 10 years ago or less, and to hospital interns and residents doing research in urology. The winner of the first prize will appear at the meeting of the American Urological Association in New Orleans, April 28-May 1, 1958. For full particulars, write to William P. Didusch, 1120 N. Charles St., Baltimore. Md. Essays must be submitted before Dec. 1, 1957.

CONFERENCES, CONGRESSES, AND CONVENTIONS

Cardiac. The Midwest Cardiac Conference will be held at Iowa State University Hospitals, Iowa City, Oct. 3-5. The co-sponsor is the Iowa Heart Association, 2100 Grand Ave., Des Moines, Iowa.

Dermatology. Advance plans for a two and a half day symposium on Current Therapy in Dermatology are in progress at the UCLA Medical Center, where University Extension will sponsor the conference next Oct. 25, 26, and 27. Three speakers of exceptional prominence in the field are Richard L. Sutton, Jr., M.D., Professor and Chairman of the Department of Dermatology, University of Kansas Medical School; Rudolph L. Baer, M.D., Associate Clinical Professor of Dermatology and Syphilology, New York University Post-Graduate Medical School, and Editor of the "Yearbook of Dermatology"; and Walter B. Shelley, M.D., Associate Professor of Dermatology, University of Pennsylvania Medical School. Requests for information should be addressed to Dr. Sternberg, University of California Extention, Medical, Los Angeles 24.

Endocrinology. The Postgraduate Assembly in Endocrinology and Metabolism will be held from Oct. 21-25 at the Medical College of Georgia.

Gastroenterology. The World Congress of Gastroenterology and American Gastroenterological Association will meet May 25-31, 1958, in Washington, D.C. Write to H. M. Pollard, M.D., University Hospital, Ann Arbor, Mich.

Internal Medicine. The International Congress of Internal Medicine will be held in Philadelphia from April 24-26, 1958. For more information, write to Edward R. Loveland, 4200 Pine St., Philadelphia.

Medical Writing. The American Medical Writers Association will meet at the Sheraton-Jefferson Hotel in St. Louis, Sept. 27-28. All members of the American Medical Writers Association and other collegiate graduates are invited to attend the meeting. The program will feature 18 medical writers and authors as speakers on Sept. 27 and a workshop on medical writing on Sept. 28. There is no charge for the meeting on Sept. 27. but a registration fee of $5 will be charged for the workshop. The Mississippi Valley Medical Society will meet at the same time and place. For further details, write to Harold Swanberg, M.D., Secretary, W.C.U. Bldg., Quincy, Ill.

National Society for Crippled Children and Adults. A series of speeches, seminars, workshops, clinics, and demonstrations will be featured at the 1957 Annual Convention of the National Society for Crippled Children and Adults, to be held Oct. 31-Nov. 2 in Chicago. Delegates from the Easter Seal societies throughout the United States, as well as lay persons interested in nonscientific aspects of the work, will participate in the meeting.

Physical Medicine and Rehabilitation. The American Congress of Physical Medicine and Rehabilitation will convene on Sept. 8-13, 1957, in Los Angeles.

COURSES

Anesthesiology. The American Society of Anesthesiologists, Inc., will present the Eighth Annual Refresher Course Program at the Hotel Statler, Los Angeles, Oct. 14-15, 1957. The course is sponsored by the Committee on Medical Schools and Postgraduate Education. For further information, write to the American Society of Anesthesiologists, 188 W. Randolph St., Chicago.

Compensation Medicine. "Medical Aspects of Workmen's Compensation" will be the subject of a one week course, Oct. 21-25, to be presented by the New York University Post-Graduate Medical School and the American Academy of Compensation Medicine. Ten sessions, a morning and afternoon session each day, will cover newer problems in compensation medicine such as "The Heart in Industry"; "Medical Problems of the Older Worker"; "Tranquilizers and the Worker"; and "Radiation." The latter sessions will encompass all the health problems of radiation not only as they affect workers dealing with radioactive materials but also as they affect average citizens subject to fallout and other radiation hazards. Applications and further information may be obtained from the Office of the Associate Dean, New York University Post-Graduate Medical School, 550 First Ave., New York 16.

Gastroenterology. A postgraduate course in gastroenterology will be given from Oct. 24-26, Hotel Somerset, Boston. For further information, write to the American College of Gastroenterology, 33 W. 60th St., New York 23.

Occupational Skin Problems. This course convenes from Oct. 28-Nov. 1. For information, write to the Institute of Industrial Health, Kettering Laboratory, Cincinnati 19.

Radiation. A one week course in radiation for industrial physicians and lawyers will be held Sept. 9-14 at the University of Cincinnati. Write to the Institute of Industrial Health, Kettering Laboratory College of Medicine, University of Cincinnati, Cincinnati 19.

FELLOWSHIPS AND SCHOLARSHIPS

Cardiovascular Diseases. Three fellowships are available each year in institutions in the central Ohio area for one, two, or three years. Stipends are from $5,000 to $7,000 per year.

University clinical fellowships at Ohio State University College of Medicine in Columbus are available to physicians who have completed clinical cardiology training and who wish to pursue further research with special attention to preparation for an academic career. Clinical fellowships are available to physicians who have completed a two year residency related to cardiovascular diseases and who wish further opportunity for study in basic sciences, clinical research, and teaching, without routine service responsibilities. Basic science fellowships are for graduate training in research in biochemistry, physiology, and other basic sciences related to cardiovascular disease. Candidate should have at least a bachelor's degree in basic science.

Fellowships may begin Jan. 1 or Oct. 1. Information may be obtained from the Executive Secretary, Central Ohio Heart Association, 50 E. Broad St., Columbus 15, Ohio.

Neuromuscular Diseases. The Sister Elizabeth Kenny Foundation has announced postdoctoral scholarships of $5,000 to $7,000 yearly for a five year period, to promote work in the field of neuromuscular diseases. Write to E. J. Huenekens, M.D., Medical Director, Elizabeth Kenny Foundation, 2400 Foshay Tower, Minneapolis 2.

Science. Citizens of the United States who have held a doctoral degree for at least five years, or who will possess such a degree by Sept. 3, 1957, are eligible for senior postdoctoral fellowships in science, to be awarded by the National Science Foundation. These awards will provide an opportunity for individuals to supplement their training. No stipend will be less than $4,000 per annum (before adjustment for supplemental income) and no award (including allowances) will be less than $2,000. Awards will be made on Oct. 17, 1957.

STUDENT LOAN FUND

Mrs. Rose Strick has established the Frank and Rose Strick Loan Fund in the amount of $11,500 at Temple University as a living monument to her husband, the late Frank Strick, an industrialist. Students in need of financial assistance will receive $500 during a single academic year. If necessary, a renewal may be arranged for another term. Students are required to repay the loan within six years.

Opportunities for Practices

An associate
at Overlook Sanitarium, New Wilmington, Pa.
Psychiatric training helpful but not essential.
Salary and maintenance.
Opportunity for private practice in addition.
Office facilities provided.
Apply: Elizabeth Veach, M.D.
Phone: New Wilmington, 5151

General practice of obstetrics and gynecology
in Amarillo, Texas.
Two women or a husband-wife medical team
associated with Dr. Ernestine Smith
and Dr. Margaret Davis.
Established practice of 16 years.
New office.
Apply: Ernestine Smith, M.D.
814 Lamar St.
Amarillo, Texas.

Combined practice
in Portland, Ore.
Two women or husband-wife medical team.
Specialties may be pediatrics, internal
medicine, general practice, or allied fields.
Established practice over 25 years.
Well conditioned to women doctors.
Will introduce to hospital and to patients.
No immediate investment required.
Apply: Jessie Laird Brodie, M.D.
732 N.W. 19th.
Portland 9, Ore.

CORRECTION

In "These Were the First" of May, 1957, page 152, the statement appeared that Johanna Thomas Zelvis Baltrusaisis was the first woman physician in Pittsburgh. Her son has advised us that his mother's name is spelled Zelwis Baltrusaitis and that she was the first woman physician in Plymouth, not Pittsburgh.

THESE WERE THE FIRST

Dr. Elizabeth Bass of Lumberton, Miss., who died in January, 1956, graduated from the Woman's Medical College of Pennsylvania in 1904 and was the first woman member of the Orleans Parish Medical Society. She was also one of the first medical women on the faculty of Tulane University School of Medicine, where she served until her retirement in 1941 as Professor of Medicine Emeritus. As Chairman of the Section of Pathology in 1939, she was the first woman officer of the Southern Medical Association. Dr. Bass was President of the AMWA (1921-1922), a member of many national and international societies, and founder of the "Women in Medicine Collection" in Matas Medical Library.

Dr. Lucy Scott Hill of Locust Dale, Va., graduated in 1924 from the University of Richmond and Medical College of Virginia and was the first woman to specialize in neuropsychiatry in New Orleans.

Dr. Margaret Ellen Holland of Newberryport, Mass., graduated in 1871 from the Woman's Medical College of Chicago and was the first woman physician to practice in Harris County, Texas. Dr. Holland was very charitable, assisted young people with their education, and reared three children belonging to her brother.

Dr. Helen M. Wallace, formerly of New York, N.Y., and Specialist in Public Health for the AMWA, is now at the University of Minnesota College of Medical Sciences, Minneapolis, where she is the first Professor of Maternal and Child Health. Dr. Wallace was formerly Chief of the Maternity and Newborn Division and Director of the Bureau for Handicapped Children in the New York City Department of Health; she was also Professor and Director of the Department of Preventive Medicine and Public Health at New York Medical College. Dr. Wallace graduated in 1937 from Columbia University College of Physicians and Surgeons.

—From the Elizabeth Bass Collection
Rudolph Matas Medical Library, Tulane University.

News of Woman's Medical College of Pennsylvania

Dr. Anna Daniels of New York was the principal speaker at a dinner given in New York for 40 members of the Parents Association of Woman's Medical College of Pennsylvania, who had come from many sections of the United States to participate in the events marking the completion of the college year and to tour the United Nations. Dr. Daniels is the mother of Dr. Dorothea D. Glass, Instructor in Preventive Medicine at the College and holder of the Lois Mattox Miller fellowship. Others on the program were: Dr. Rita E. Scott, President of the College's Alumnae Association, and Dr. Rose Mikelberg, President of the Parents Association and Clinical Assistant Professor of Pediatrics at the College.

Among the special guests at the dinner were: Dr. Frieda Baumann, Editor of the *Journal of the American Medical Women's Association*, Dr. Hannah E. Longshore, Professor of Medicine, Dr. Katharine R. Boucot, Professor of Preventive Medicine and Clinical Professor of Medicine, and Dr. Jean Crump, Professor of Pediatrics at Woman's Medical College.

Dr. Sophie Rabinoff received the Annual Alumnae Achievement award. Dr. Rabinoff, a member of the class of 1913 at Woman's Medical College, is Professor of Public Health and Industrial Medicine at the New York Medical College.

Dr. Katharine R. Boucot was the main speaker at the graduation exercises of the largest class of graduates in the history of the College's School of Nursing. Mrs. John B. Kelly, Chairman of the School of Nursing Committee and Vice-President of the Board of Corporators of the College, presented the prizes to the outstanding students.

Dr. Alma Dea Morani, a plastic surgeon of Philadelphia and faculty member of Woman's Medical College, was installed as President of the Alumnae Association. Other officers are: Dr. Anne H. Pike, class of 1946, President-Elect; Dr. Eloise Lemon, Treasurer; Dr. Anne Gray Taylor, Achievement Award Committee; Dr. Emily Loeffler, Membership Committee; Dr. Jean Crump, Maine Camp Committee; Dr. Grace R. Nachod, Publications Committee; and Dr. Elizabeth S. Waugh, Chairman of the Nominating Committee.

Dr. Mary M. Spears, class of 1916, formerly Professor and now Emeritus Professor of Gastroenterology and Proctology, was awarded the degree of doctor of medical science for her outstanding career in the field of medicine. Other honorary degrees conferred were degrees of doctor of humane letters on Mrs. Albert D. Lasker, who, with her late husband, founded the Mary and Albert Lasker Foundation, which provides money for medical research, and the degree of doctor of social science on Mrs. Alice K. Leopold, Director of the Women's Bureau, Department of Labor, and Assistant to the Secretary of Labor for Women's Affairs. Mrs. Leopold was the commencement speaker. Her topic was "Medicine, the Great Ambassador."

Dr. Marion Spencer Fay, Dean of Woman's Medical College, received an honorary doctor of medical science degree from Beaver College at their 104th commencement exercises on June 9.

Dr. Raymon Kistler, President of Beaver College, said: ". . . you used your talent as a teacher in Colorado, in Connecticut and in Texas, coming to the Woman's Medical College in 1935. Since 1943, you have served as Dean of this outstanding Institution for the preparation of women in the field of medicine. We honor you because of your membership in all the leading Societies in your field, because you hold honorary degrees from the Women's Medical College and Temple University, and because you have been selected as a Distinguished Daughter of Pennsylvania."

News of Women in Medicine

Dr. Mary Bacon of Cumberland County is serving as an alternate member of the Nominating Committee of the New Jersey Medical Society. She is also a member of Reference Committee "A" for the state society.

Dr. Mary R. Boyd has been appointed Health Commissioner of Clinton County, Wilmington, Ohio.

Dr. Alice Coughlan has retired after serving 42 years as School Physician of the Orange, N.J., public schools and was honored with a luncheon by her many friends and colleagues. Dr. Coughlan retired from private practice as a gynecologist in 1948. A summer in Maine, a world cruise, and then back to Orange are her immediate plans for the future. Dr. Coughlan determined early in life to enter medicine but was denied this privilege because her father did not approve of college for women. When she received an inheritance from her father, she spent it on medical education, graduating in 1914. When she came to New Jersey she scored 100 per cent, an unheard of achievement, in the state medical jurisprudence examination. Dr. Coughlan has been an active member of the AMWA since 1931.

Dr. Maria Fleischel of New York received first prize in the seascape and landscape section of the nineteenth annual exhibition of the American Physicians Art Association, and Dr. Winifred Ingusall of Laramie, Wyo., took top honors in the portrait division.

Dr. Barbara Ann Higbie of East Orange, N.J., was the recipient of the 1957 Mead Johnson award of the American Academy of General Practice. Dr. Higbie, who has just completed her residency in general practice at the Hunterton Medical Center in Flemington, N.J., is the first woman to receive the $1,000 scholarship from the American Academy of General Practice and the first New Jersey physician to receive the award. She will begin a residency in pediatrics at the Philadelphia Children's Hospital on July 1. Dr. Higbie is a native of New Jersey and a graduate of Woman's Medical College of Pennsylvania. The award is made to encourage more physicians to take graduate training in general practice.

A special feature of the 1957 meeting of the Medical Society of New Jersey was the first bestowal of the Society's Golden Merit award, a citation conferred upon members who have completed 50 years of medical practice. Among the first recipients were: Dr. Harriet L. Knox, Hackensack; Dr. Frances B. Tyson, Leonia, who was elected emeritus member of the AMWA at the 1957 Annual Meeting; Dr. Emma Weeks Metzer, Riverside; Dr. Stella S. Bradford, Montclair; Dr. Ellen C. Potter, Trenton; and Dr. Mary B. Carr, Dr. J. Eugenia Jaques, and Dr. Emilie V. Rundlett, all of Jersey City.

Dr. Hilda H. Kroeger, Administrator, Elizabeth Steel Magee Hospital, Pittsburgh, and Dr. Martha O'Malley, Director, Division of Hospital and Institutional Services, Indiana State Board of Health, have been elected to two year terms as members of the Board of Directors of the American Committee on Maternal Welfare, Inc., and Dr. Goldie B. Corneliuson, of Lansing, Mich., Director, Division of Maternal and Child Health, Michigan Department of Health, was elected for a three year term.

Dr. Esther Pohl Lovejoy, author of the new book "Women Doctors of the World," was the subject of a picture taken at the 1957 Annual Meeting of the AMWA and published in *Scope Weekly*. The animation evident in the picture was induced by only one thing, the overwhelming interest of Dr. Lovejoy, throughout her long and her full career, in women doctors and in their accomplishments.

Dr. Delia A. Lynch of Omaha, Neb., suffered a heart attack recently and has had to curtail many of her former activities. Dr. Lynch is well known to many members of the Association for her faithful attendance at national meetings and for her valuable gift of the picture "Dante and Beatrice," which will hang in the American Medical Women's Library at the Woman's Medical College of Pennsylvania upon completion of this wing to the College buildings.

Dr. Alice E. Palmer has been elected Recorder for the Detroit Dermatological Society.

ALBUM OF WOMEN IN MEDICINE

RUTH RENTER DARROW, M.D.

1895-1956

Ruth Renter Darrow was born on Sept. 25, 1895, a native of Cleveland, and died at Women and Children's Hospital, Chicago, of cerebral hemorrhage on April 23, 1956. She had been a member of the staff of that hospital since 1934 and served for one year as its president. A graduate of Flora Mather College, Western Reserve University, and Rush Medical School, she served her internship at Michael Reese Hospital. An outstanding scholar, she was a member of the Alpha Omega Alpha and the Sigma Xi societies.

Dr. Darrow was intensely interested in the study of erythroblastosis. This was stimulated by the death of her son Alan in 1935 of erythroblastosis and led to her excellent article "Icterus Gravis (Erythroblastosis) Neonatorum: Examination of Etiologic Considerations," published in 1938 in the *Archives of Pathology*. In this article she proposed the antigen - antibody theory as the reaction best explaining all aspects of erythroblastosis. Several articles were published later relating to the Rh factor, and one, "Specificity of Fetal and of Adult Human Hemoglobin Precipitins," was undertaken to determine whether hemoglobin in the newborn infant differs as an antigen from that in the human adult.

It is perhaps needless to say that she was a person of exceptional mental ability and that she had a remarkable faculty for pursuing an idea to its conclusion. Her intensity of interest in the Rh factor carried over to her associates and friends. In addition to a very active medical practice and a busy home life, she found time in the evenings and early morning hours to do research work in the field of Rh. However, in recent years it was necessary for her to put aside most of her research work and some of her practice in order to care for her invalid mother, but she planned a return to her work as soon as possible. She was a proponent of the use of Rh-positive blood in the treatment of erythroblastosis "because of its sparing action on the infant's red blood cells, its greater desensitizing effect, and its availability from the patient's family."

Dr. Darrow is survived by her husband, Dr. Chester W. Darrow, who is an associate professor in the Department of Physiology at the University of Illinois College of Medicine, and by three daughters: Mrs. Robin Oggins, Mrs. Roland Grybeck, and Gail, a second year high school student.

A devoted physician, Dr. Darrow took great pride in her patients and is remembered by them not only as a doctor but also as a friend. Her death is a serious loss to the medical profession, to her community, and to her family.

—Irene Shmigelsky, M.D.

Book Notices

Editor's Note: These reviews represent the individual opinions of the reviewers and not necessarily those of the members of the Editorial Board of the JOURNAL.

ROENTGEN SIGNS IN CLINICAL DIAGNOSIS. By Isidore Meschan, M.A., M.D., Professor and Director of the Department of Radiology at the Bowman Gray School of Medicine of Wake College, Winston-Salem, North Carolina. Pp. 1,058, with 2,216 illustrations and 760 figures. Price $20.00. W. B. Saunders Company, Philadelphia, 1956.

The author goes to considerable length to explain in a very clear fashion why the x-ray appearance is as it is. He explains changes in structures which account for the x-ray appearance and he classifies all these findings in terms of size, architecture, density, anatomy, and physiology. The book is designed primarily for the radiologist, but, because of its clarity, it will be very useful for anyone else using radiologic techniques or having to pass opinions on films.

The author discusses each body area in the light of the technical aspects of x-ray diagnosis. He discusses the anatomy of the patient, the particular exposures to be used, the types of media to be used, and what is best in order to obtain the best results in any given situation. The anatomy of the area is discussed in detail, and one of the most helpful parts of the book is the discussion of the routine examination of any given area which is to be examined by x-ray.

One of the outstanding features of the book is Dr. Meschan's summation, which is divided into four main features. The first is the x-ray appearance or the lesion; the second is the altered physiology of the lesion as it affects the x-ray appearance; the third is the significance of all these findings; and the fourth is the differential diagnosis of lesions which may be readily confused with other lesions.

The make-up of this book is attractive and pleasing and the book is recommended without reservation.

—Lois I. King, M.D.

HUMAN PROBLEMS OF A STATE MENTAL HOSPITAL. By Ivan Belknap, Ph.D., Associate Professor of Sociology, The University of Texas. Pp. 277. Price $5.50. The Blakiston Division, McGraw-Hill Book Company, Inc., New York, 1956.

This book is a thorough and penetrating study of the history and development of the state hospital in the United States, based on a three year study of "Southern State Hospital," a mythical average state hospital. Dr. Belknap has investigated meticulously every detail of the physical structure and personnel organization. He has shown clearly all the defects in both categories and has come up with rather startling statistics and ideas. The state hospital, as it exists, appears to be a prime factor in the failure of the present-day treatment of the mentally ill who are sent to such an institution.

In conclusion, Dr. Belknap offers several concrete ideas for improvement. Building larger hospitals and thereby increasing the burden on the overworked physicians, the nurses, social workers, and the poorly paid attendants would only make matters worse. The first step would be to decentralize the large hospital into several community hospitals and mental hygiene clinics. There should be a revamping of the present organization of personnel and an adequate compensation to attract qualified physicians, nurses, social workers, psychologists, and attendants.

This book should be of great interest and importance to any worker in the field of psychiatry and to any physician who is associated with the treatment of the mentally ill.

—Hilda Ratner, M.D.

THE INTERPRETATION OF THE UNIPOLAR ELECTROCARDIOGRAM. By Gordon B. Myers, M.D., Professor of Medicine, Wayne University College of Medicine; Head, Department of Medicine, City of Detroit Receiving Hospital. Pp. 164, illustrated. Price $4.75. The C. V. Mosby Company, St. Louis, 1956.

This is an excellent, useful text on interpretation of the unipolar electrocardiogram. Dr. Myers has given us a simple, direct, and readable account which has basis in the experimental work he has done. The correlation between the electrocardiogram in life and the post-mortem examination has been an important contribution to our knowledge of localization of myocardial infarction and has been included here.

For general use, particularly in the discussion of the arrhythmias, more electrocardiogram illustrations would lend emphasis and add clarity to the discussion.

This is a book which should have a place in every hospital library for reference.

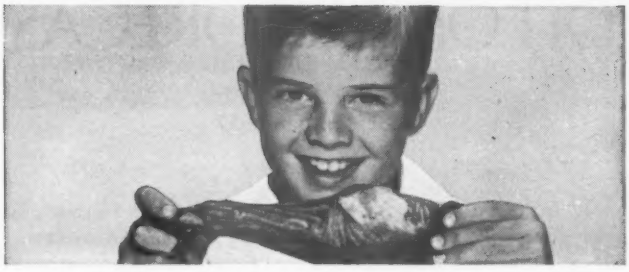
—Frances Bailen-Rose, M.D.

THE FOUNDERS OF NEUROLOGY. One Hundred and Thirty-Three Biographical Sketches. Prepared for the Fourth International Neurological Congress in Paris by 84 authors. Edited by Webb Haymaker, M.D., Chief, Neuropathology Section, Armed Forces Institute of Pathology, Washington, D.C. Pp. 479, illustrated. Price $10.50. Charles C Thomas, Springfield, Ill., 1956.

The founders of neurology are divided into neuroanatomists, neurophysiologists, neuropathologists, clinical neurologists, and neurosurgeons, an arrangement that makes it easy for the subspecialist to review his own field but that makes it a little disorderly for the reader who would like to know something of the general history of this lofty branch of medicine.

The quality of writing varies, but, in general, good editing has produced remarkable uniformity. Opening sentences vary form "Edinger was born in Worms on the Rhine" to "Whether the lines of progress in electrophysiology be traced backward over a century," but pertinent facts are arranged in orderly fashion. Both as a reference and for good reading, this small volume is useful to all.

—Margaret H. Edwards, M.D.



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EDITORIAL FORECAST

September 1957

Dr. Hulda E. Thelander is the Guest Editor for the special Children's Hospital, San Francisco, issue of the JOURNAL. She has collected informative and interesting articles and has chosen Dr. Mary Glover, beloved physician of San Francisco who died recently, for the Album of Women in Medicine.

"Long-Continued Adrenal Hormone Therapy in Childhood Nephrosis," by Carolyn F. Piel, M.D., and Gordan F. William, M.D.

"Plasma Thromboplastin Component (PTC) Deficiency," by Paul M. Aggeler, M.D., M. Silvija Hoag, M.D., and Ralph O. Wallerstein, M.D.

"Presentation of a Pneumotachograph with Reference to Respiratory Distress in the Newborn Infant," by Pieter A. deVries, M.D., Robert W. Vreeland, B.S., and Carter Collins, M.A.

"Tumors in Childhood and the Role of a Pediatric Tumor Board," by Joseph H. Kushner, M.D., Evelyn Siris, M.D., and Robert S. Sherman, Jr., M.D.

"Pediatrics Residency" (Letter to an Intern), by Dorothy A. English, M.D.

"A Resident Training Program," by H. E. Thelander, M.A., M.D.

Also included in the September JOURNAL will be the summarized minutes of the Annual Meeting, held May 30, 31, June 1, and 2, 1957, in New York City.

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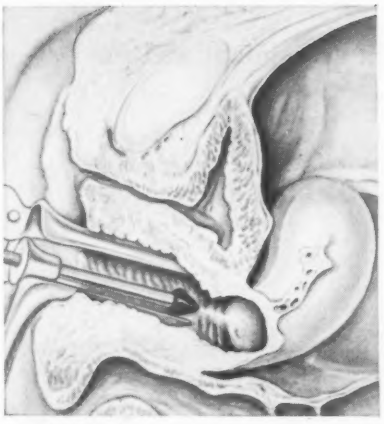

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*Williamson, P.: Trichomonad Infestation, M. Times *84*:929 (Sept.) 1956.

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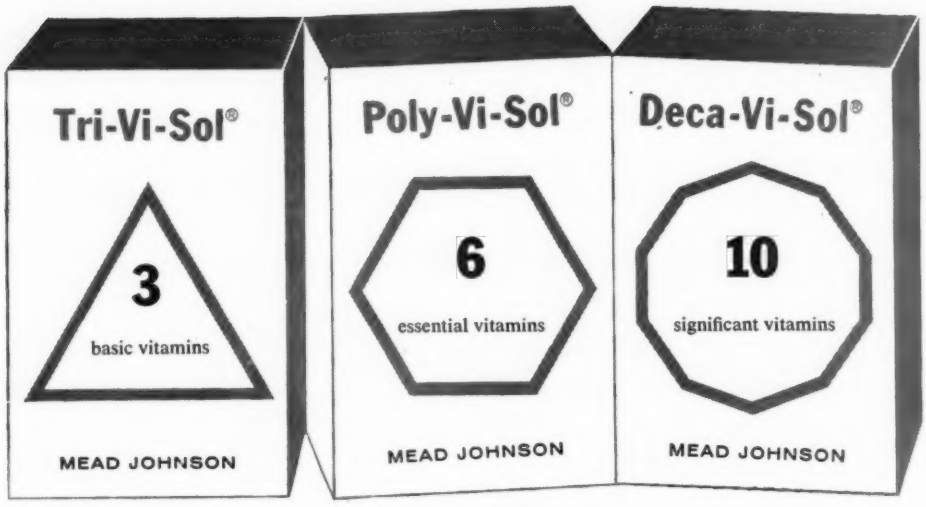

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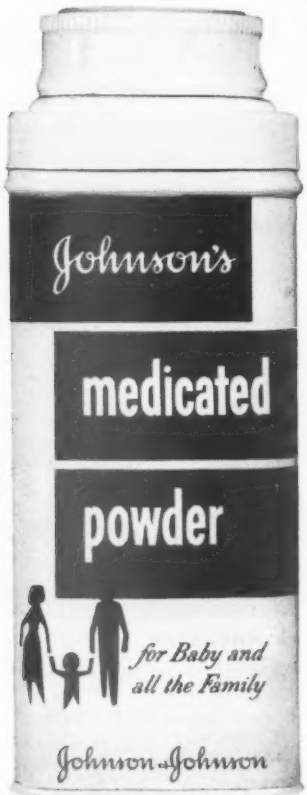

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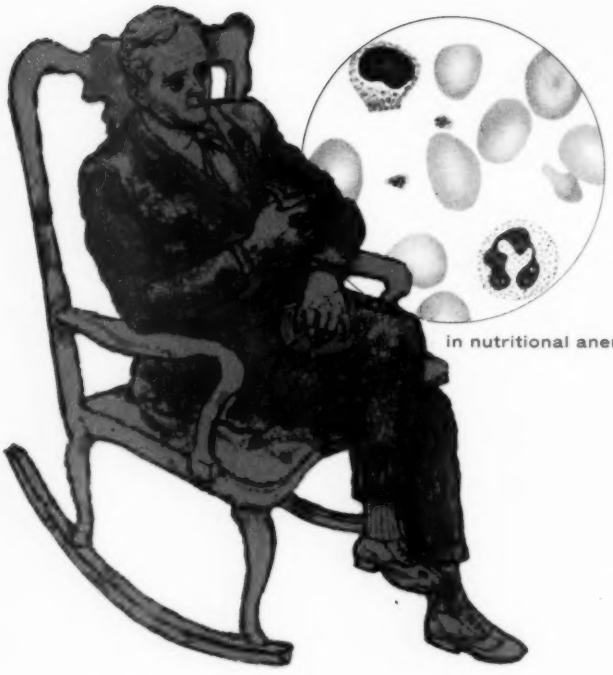

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